"ASSESSMENT OF RIGHT VENTRICULAR FUNCTION BY ECHOCARDIOGRAPHY IN PATIENTS WITH INFERIOR WALL MYOCARDIAL INFARCTION"

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CHENNAI – 600 003

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CHENNAI – 600 032

AUGUST 2009

"Learn to heal"

CERTIFICATE

This is to certify that the dissertation entitled **"ASSESSMENT OF RIGHT VENTRICULAR FUNCTION BY ECHOCARDIOGRAPHY IN PATIENTS WITH INFERIOR WALL MYOCARDIAL INFARCTION"** is the bonafide original work of **DR.S.RAGHOTHAMAN** in partial fulfillment of the requirements for D.M. Branch-II (CARDIOLOGY) examination of THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY to be held in August 2009. The period of post-graduate study and training was from July 2006 to July 2009.

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DECLARATION

I **Dr. S.RAGHOTHAMAN,** solemnly declare that this dissertation entitled, **"ASSESSMENT OF RIGHT VENTRICULAR FUNCTION BY ECHOCARDIOGRAPHY IN PATIENTS WITH INFERIOR WALL MYOCARDIAL INFARCTION"** is a bonafide work done by me at the department of Cardiology, Madras Medical College and Government General Hospital during the period 2006 – 2009 under the guidance and supervision of the Professor and Head of the department of Cardiology of Madras Medical College and Government General Hospital, Professor **R.Subramanian M.D.D.M.** This dissertation is submitted to The Tamil Nadu Dr.M.G.R Medical University, towards partial fulfillment of requirement for the award of **D.M. Degree (Branch-II) in Cardiology.**

Place : Chennai

Date: 27.05.2009

Dr.S.RAGHOTHAMAN

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Last but not the least I thank all my patients for their kind cooperation.

CONTENTS

INTRODUCTION

Inferior wall myocardial infarction in complicated by right ventricular infarction (RVMI) in as much as 50% of cases. In a series by Anderson et al in 1989, the incidence of RVMI in patients with inferior wall myocardial infarction was between 10 – 50%. Isolated infarction of the right ventricle is extremely rare.

Although right ventricular infarction is clinically evident in a sizable number of cases, the incidence is considerably less than that found at autopsy.[1] A major reason for the discrepancy is the difficulty in establishing the presence of RVMI in living patients. Additionally, right ventricular dysfunction and stunning frequently is of a transient nature, such that estimation of its true incidence is even more difficult. Criteria have been set for to diagnose RVMI; but, even when strictly employed, the criteria lead to underestimation of the true incidence of right ventricular infarction.

In patients with RVMI, the risk of death in the hospital is high and major complications are greater. Right ventricular infarction contributes markedly to hemodynamic instability, atrio-ventricular conduction blocks, and in-hospital mortality in patients with inferior wall myocardial infarction. Systolic right ventricular function is an important predictor in the course of myocardial infarction. Despite this initial observation nearly two decades ago, this condition has received little clinical attention until recent years.

As compared with all clinical variables available at the time of admission, RVMI is associated with

a relative risk of in-hospital mortality of 7.7 (95% CI) and a risk of major in-hospital complications of 4.7 (95% CI) [2]. The potential hemodynamic derangements associated with right ventricular infarction render the afflicted patient unusually sensitive to diminished preload and loss of atrio ventricular synchrony. These two circumstances can result in a severe decrease in right and, secondarily left ventricular output. [3]

According to the Shock Trial Registry[38], despite the younger age, lower rate of anterior MI, and higher prevalence of single vessel coronary disease of RV compared with LV shock patients, and their similar benefit from revascularization, mortality is unexpectedly high in patients with predominant RV shock and similar to patients with LV shock.

Cardiogenic shock and the requirement for temporary transvenous cardiac pacing are more common in patients with right ventricular dilatation. Furthermore, by implying multivessel coronary artery disease, the presence of right ventricular dysfunction carries an adverse prognosis irrespective of infarct location. The demonstration of right ventricular dysfunction is important because it is often associated with a distinct clinical syndrome requiring specific management. In the presence of low cardiac output volume loading to restore left ventricular filling pressure is required. Inappropriate vasodilator and/or diuretic therapy may prove fatal. Information about right ventricular function can be applied in thrombolysis decision making when a relative contraindication is present. In fact, the findings of one study suggested that patients with inferior myocardial infarction derived no benefit from thrombolysis in the absence of right ventricular involvement. Interest in recognizing right ventricular infarction non-invasively has grown because of the therapeutic implications of distinguishing patients with right ventricular dysfunction from those without right ventricular dysfunction[4].

For early diagnosis, electrocardiography and two dimensional echocardiography are used, but, these methods are occasionally insufficient. There are a lot of problems in assessing the function of the right ventricle because,

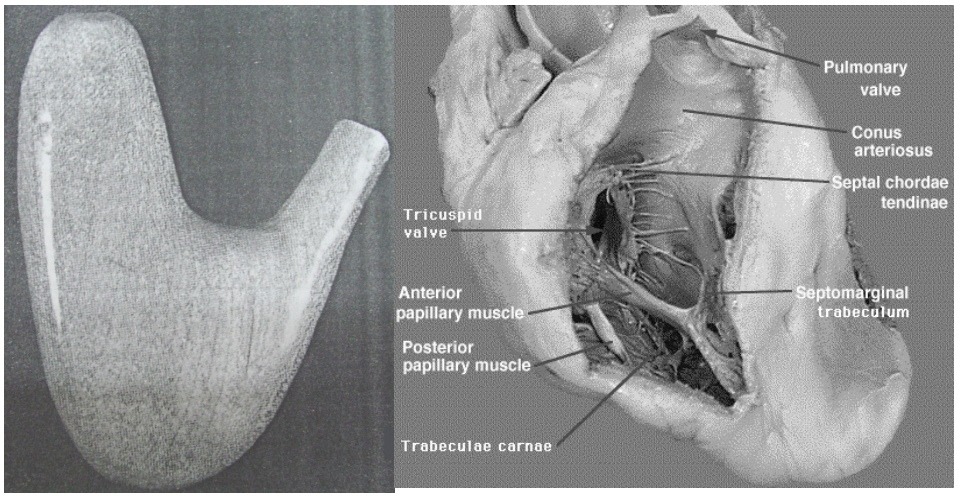
a. Right ventricle is crescentric and truncated into separate inflow and outflow portions (Pic 1).

b. Endocardial boundary is not accurately localizable due to variable trabeculation pattern.

c. Right ventricle has a complex wall motion with the inflow portion contracting before the trabecular and outflow portion.

d. Right ventricle is located behind the sternum and hence is inaccessible to imaging.

e. Right ventricle poorly approximates to any geometrical model.



Picture 1. The complex anatomy of the right ventricle.

Also, in the vast majority of patients with right ventricular infarction, the wall motion abnormalities initially manifest on echocardiography reverse within 3 months[49]

To allow optimal patient management an ideal measure of right ventricular performance should give an accurate assessment of function, be practicable in everyday clinical use and provide accurate prognostic information.

Use of echocardiography in the assessment of right ventricular function:

Echocardiography has been and continues to be the most commonly used and most readily available investigation modality for the assessment of right ventricular function. Various echo parameters have been in use, like the tricuspid annular motion, right ventricular dimensions, myocardial performance index, tissue doppler imaging etc. Each of them lack sensitivity and specificity

when applied individually; they have their own drawbacks, and that's why the combination of various parameters always give a better and more accurate estimate of right ventricular systolic function, especially in the setting of acute myocardial infarction. Apart from these, newer methods are currently under investigation like the 3D echocardiography, intra cardiac echocardiography, velocity vector imaging, strain & strain rate imaging, colour kinesis and automated border detection methods. Echocardiography will continue to be the first line investigation of choice for the assessment of right ventricular function for the time to come.

Other non-invasive assessment of right ventricular function:

The various methods of assessing right ventricular function non-invasively other than echocardiography include

a. ***Radionuclide angiography:*** Both gated first-pass and equilibrium radionuclide angiography are useful, but, radionuclide techniques are limited by inter-observer and inter-study variability, radiation, high cost, time for acquisition and processing and inability to separate the atrium and the ventricle.

b. ***Cardiac computed tomography*****:** Ultra-fast cine computed tomography provides an excellent avenue for assessment of cardiac morphology and comparison to actual RV volumes are shown to be very accurate. It permits high spatial, temporal and contrast resolution for delineation of Endocardial and epicardial borders without any assumptions of the RV geometry as commonly done by other methods.

c. ***Cardiac magnetic resonance:*** It is the non-invasive gold standard for the estimation of right ventricular volume and wall mass. Magnetic resonance imaging provides a method of accurately visualizing the complex internal architecture of the right ventricular cavity[20]. Contrast is not needed for its use. No geometric assumptions are made. Unfortunately, it is not widely available, data is lacking on prognostic value and serial assessment is not feasible.

Invasive assessment of right ventricular function:

Angiocardiographic estimation of right ventricular volume is invasive and is not widely performed. Accurate measurements are reliant on the selection of appropriate orthogonal planes and accurate border tracing. A large variety of geometric figures have been used and many overestimate true volume probably due to problems with inclusion of right ventricular myocardium in heavily trabeculated regions of the ventricle. Both first pass and equilibrium techniques have been applied. Each has limitations and it is unlikely that the favourable mean inter-observer and inter-study variabilities for the estimation of left ventricular ejection fraction are applicable to the right ventricle.

To conclude, echocardiography provides a readily accessible tool for the evaluation of right ventricular function. It remains the first line investigation because of its ability to provide comprehensive information on right ventricular size, structure and function. Recent developments in ultrasound technology have overcome the limitations of simple M-mode and two-dimensional imaging and facilitated more accurate monitoring of disease progression. Competition from quantitative, highly reproducible techniques is emerging. Magnetic resonance imaging has become the gold standard technique for evaluation of right ventricular volume and function but a balance needs to be struck between techniques which are available, accurately reflect right ventricular performance and are of proven prognostic value.

AIM OF THE STUDY

- To compare various echo methodologies in assessing Right Ventricular function in the setting of Inferior wall myocardial infarction with and without right ventricular involvement.
- To assess RV function by echocardiography done at different time intervals after the onset of myocardial infarction.
- To compare RV function in patients who are thrombolysed & those not thrombolysed.
- To compare RV function in males & females.
- To compare RV function in different age groups.
- To assess occult RV dysfunction by echocardiography in patients without Right Ventricular Myocardial Infarction on ECG.

REVIEW OF LITERATURE

Clinical features of right ventricular infarction:

- A right ventricular infarct should be considered in all patients who present with an acute inferior wall myocardial infarction, especially in the setting of a low cardiac output.
- Patients may describe symptoms consistent with hypotension and they are markedly sensitive to preload reducing agents such as nitrates, morphine or diuretics[47].
- The classic triad of distended neck veins, clear lung fields and hypotension occurs in 25% of cases[48].
- On hemodynamic monitoring, disproportionate elevation of right sided filling pressures compared with left sided hemodynamics represents the hallmark of right ventricular infarction[32].

Sequelae of right ventricular myocardial infarction:

RV systolic dysfunction	➡↓ RV cardiac output	➡↓ LV filling
RV dilatation	➡ Septal shift toward LV	➡↓ LV compliance
	➡↑ Pericardial pressure	➡↓ LV compliance
	➡ Change in LV geometry	➡↓ LV systolic function
Atrioventricular dyssynchrony	➡ Loss of atrial contribution to ventricular filling	➡↓ LV cardiac output
Concomitant LV infarction	➡↓ LV systolic function	➡↓LV cardiac output

Complications of Right ventricular myocardial infarction:

In a study by Garty et al[50], complications occurred in 70% of cases of RVMI especially in the first 24 hours, and the commonest complication was conduction abnormalities. Hypotension occurred in 15 of 46 patients (33%) with RV infarction. The prevalence of second- and third-degree AV block was 24 of 46 patients (58%). Prevalence of second- or third-degree AV block occurred in 3 of 4 patients, or 75%. Other complications include RV dysfunction leading to poor cardiac output, tricuspid regurgitation, and pulmonary hypertension. Mortality is increased in the presence of right ventricular myocardial infarction, especially in the presence of cardiogenic shock and pulmonary hypertension.

Electrocardiography:

Diagnosis of IWMI and localization of the artery involved:

	Proximal RCA	Distal RCA	Left Circumflex
ST elevation	III > II	III > II	II > II
V4R	ST elevation, T upright	ST baseline, T upright	ST depression, T inversion
L-I, aVL	ST depression		ST elevation
V1, V2	ST elevation	ST depression	-
ST dep V3 / ST elevation L-III	< 0.5	0.5 – 1.2	> 1.2
ST elevation V5,V6	-	-	+

Table 1. Infarct artery localization by electrocardiography

This method of localization of the infarct related artery by ECG has 90% sensitivity and 71% specificity.

Kabaksi et al[45] concluded that ST elevation in L–III > L-II and ST depression in L–I > aVL is 64% sensitive, but 100% specific for locating RCA as the culprit artery.

The diagnosis of right ventricular infarction in the presence of inferior wall myocardial infarction is made on the ECG based on the following findings:

- ST elevation in V4R >1mm. This is 88% sensitive and 78% specific[46]

- Usually, ST depression in V2 is at least 50% of ST elevation in aVF. If it is <50%, it denotes right ventricular infarction
- ST elevation in V1 with ST depression in V2.

The problems with using right sided chest leads for the diagnosis for right ventricular myocardial infarction are that the specificity is low; ST elevation can be present in V4R in 18% of patients even in the absence of RVMI[46], especially in the presence of any heart disease that may induce ST segment elevation in lead V1, such as pericardial disease, acute pulmonary embolism, LAFB, and acute AWMI. Also this finding of ST elevation in right sided leads disappears after 10 hours in at least 50% of cases. .

Echocardiography of the Right Ventricle:

Echocardiographic assessment of the right ventricle involves the use of M-mode, 2D, colour doppler, pulse wave doppler, continuous wave doppler and tissue doppler imaging.

1. ***M-mode:*** Assessment of RV systolic function by qualitative and semi-quantitative methods include

-RV dimensions

-RV area & shape

-Wall thickness

-Segmental motion analysis of RV free wall

-Pattern of ventricular septal motion

-Eccentricity index

2. ***Assessment of RV systolic function by quantitative methods:***

-RV volume

-RV ejection fraction

-RV dp/dt

-Pulmonary artery systolic pressure

3. ***Doppler tissue imaging***

4. ***Myocardial performance index***

Early Methods – M-mode:

Attempts to measure right ventricular dimensions echocardiographically were made soon after the development of ultrasound. Louridas[6] showed that M-mode echocardiography of right ventricular internal dimensions was significantly different between normal individuals and patients with cor pulmonale. M-mode measurements of right ventricular dimensions are, however, frequently not feasible and limitations became apparent, in particular the fact that the dimensions were so heavily dependent on the angle traversed by the ultrasound beam across the ventricle.

Two-dimensional Imaging:

Measurement of long and short-axis dimensions of right heart casts by two-dimensional imaging was quickly shown to be more accurate than M-mode techniques[7]. Two-dimensional imaging allows estimation of right ventricular dimensions, shape and wall thickness. In normal subjects, feasibility for assessment of right ventricular dimensions is high, with low inter and intraobserver variability. Developments in ultrasound technology, such as harmonic imaging and improved endocardial border definition, have further improved feasibility. Calculation of right ventricular area based on single plane echocardiographic methods correlate with right ventricular ejection fraction but assume a constant relationship between the dimensions of the ventricle in two planes. Estimation of right ventricular volume is the theoretical ideal but is based on the biplane approach requiring perpendicular echocardiographic planes, and standardization of views is difficult. A combination of apical four-chamber and subcostal right ventricular outflow tract views is the most used [8]. Many geometric models have been applied to the different ventricular views to estimate right ventricular volume. Simpson's method, which calculates volume based on a summation of the volume of individual slices, appears to

be the best approach.

Doppler Echocardiography:

With the introduction of Doppler echocardiography other parameters have been examined as measures of right ventricular performance. The widest application of Doppler to the right heart has been to estimate pulmonary artery systolic pressure by measuring the peak velocity of a tricuspid regurgitant jet and applying the modified Bernoulli equation. Although the relationship between pulmonary artery pressure and right ventricular function is complex and remains to be adequately elucidated, Doppler ultrasound is reliable in the detection of tricuspid regurgitation and a clearly defined continuous wave Doppler profile can be recorded in most patients. The application of echocardiographic contrast agents has further improved feasibility. The tricuspid regurgitant profile on continuous wave Doppler can also be used to derive the rate of right ventricular pressure rise in early systole which is a measure of right ventricular systolic function although measurements are influenced by pulmonary artery pressure. Systolic time intervals such as duration of the right ventricular pre-ejection period and ratio of the preejection period to the right ventricular ejection time have been investigated and found to correlate with similar intervals derived from two-dimensional images and right ventricular ejection fraction[9-11].

Tricuspid Annular Peak Systolic Excursion (TAPSE):

As a non-volumetric ventricular parameter, tricuspid annular peak systolic excursion (TAPSE) has provided an alternative for estimating global systolic RV function, whereby EF has been found to be approximated by 3 times TAPSE in mm as obtained from the apical 4-chamber view [23]. The one-dimensionality of TAPSE characterizing RV long axis function is at the same time its advantage and limitation, since sources of measurement error introduced by echocardiographic RV area determination are prevented, but on the other hand, global function is extrapolated from a single variable for regional systolic RV function. Since the tricuspid valve moves toward the RV apex during ventricular systole as

lengthwise shortening of both the interventricular septum and RV free wall, it is intuitively evident that TAPSE or TAPSE per time must be related to RV EF. It is imaginable that factors such as body surface area of the individual, tethering of the septal tricuspid but also mitral annulus (and thus LV function), the degree of LV myocardial mass, the presence of pulmonary artery hypertension or atrial fibrillation and other factors influence TAPSE or TAPSE per time, and thus indirectly RV EF. Analysis of systolic RV long axis function using pure velocity data has the principle advantage of practicability for the following reasons: an apical 4-chamber window is available in the vast majority of patients, tricuspid annular free wall velocity data can be obtained using a simple M-mode measurement even without TDI software, there is no data post processing required as with strain rate and strain imaging. Kaul et al. demonstrated a simple method to measure the systolic excursion of the tricuspid ring from base to apex. This motion correlated with radionuclide derived ejection fraction. Recently, Meluzin et al. showed by using Doppler tissue imaging, that the peak systolic velocity of right ventricular myocardial free wall correlated to ejection fraction. The major advantage of these measurements is the simplicity and the high reproducibility. However, the use of peak systolic velocity in this respect is limited by being both load and heart rate dependent as well as being insensitive to alteration in the inotropic state. Another limitation of TAPSE is that it represents myocardial motion only in the longitudinal direction, and not in the circumferential plane.

Doppler Index of Myocardial Performance:

There has been considerable recent interest in the application of this index initially described by Tei[12] to the right ventricle. The index of myocardial performance encompasses important energy dependent periods of systolic contraction, ejection and diastolic relaxation. The use of Doppler offers the possibility of high feasibility in patients with poor image quality and accurate characterization of function in the context of complex chamber geometry. Calculation of the index of myocardial performance is based on measurement of Doppler derived time intervals. In the case of the right ventricle relevant intervals are measured from the tricuspid inflow and pulmonary ejection profiles in

the apical four chamber and parasternal short axis views respectively. The isovolumic contraction time increases in systolic dysfunction and the right ventricular ejection time decreases. Most abnormalities of diastolic function are manifested in an abnormally slow rate of pressure decline. These changes are reflected in a prolongation of the isovolumic relaxation time. As global myocardial dysfunction progresses, therefore, the value for the index of myocardial performance increases, due to changes in all three time interval components used for its calculation. The clinical utility of the index of myocardial performance has been validated in congenital heart disease, primary pulmonary hypertension and chronic respiratory disease. It has been shown to have prognostic relevance[13]. The potential problems with the index of myocardial performance are that it is invalidated by heart block and arrhythmias. Primary valvular diseases also lead to problems in interpretation. Pre-existing lung disease, Primary pulmonary hypertension and acute cor pulmonale due to pulmonary embolism can increase MPI and will have to be excluded clinically. The effects of changes in loading conditions on the index of myocardial performance are still the subject of debate. It is particularly important to validate local values for the index of myocardial performance before applying them in clinical practice.

Tissue doppler index:

Doppler tissue imaging has previously been suggested to be a reliable tool for the evaluation of regional myocardial function throughout the cardiac cycle. Myocardial motion during the isovolumic periods, which is easily detected by Doppler tissue imaging has recently attracted much interest. The duration and the peak velocity for the isovolumic contraction phase are proposed to reflect myocardial function and correlate to increased right ventricular end diastolic pressure. Recently, Vogel et al. demonstrated by using Doppler tissue imaging, that acceleration of isovolumic contraction velocity is a reliable measurement of right ventricular contractility. Furthermore, this parameter is not affected by loading conditions and is therefore useful as an index of RV function[29].

MATERIAL AND METHODS.

This study was performed in the Department of Cardiology, Government General Hospital, Chennai, during the year 2006 – 2009. The study is a prospective case control study involving 124 patients.

STUDY GROUP SELECTION:

Ethical committee clearance was obtained to conduct the study in our hospital.

All subjects provided written informed consent to participate in the study before inclusion.

Inclusion Criteria:

1. IWMI with and without RVMI on ECG.
2. First MI (MI diagnosed by history, ECG & enzymes).
3. Within 48 hours of onset of chest pain.
4. Any age group
5. Both sexes
6. Both thrombolysed & not thrombolysed patients.

Exclusion Criteria:

1. COPD/PPH/Cor pulmonale
2. Valvular HD/Congenital HD.
3. Previous MI.
4. Complete heart block, Arrhythmias during echo.
5. DCM/LHF of any cause
6. Technically inadequate echo.

Patient characteristics:

The study population included 124 patients (78 male and 26 female) admitted to the Coronary Care Unit of Govt. General Hospital, Chennai – 3, with a first Q wave acute inferior myocardial infarction with or without RVMI.

The patients were divided into two groups:

Group 1 – Patients with IWMI with RVMI (n = 51)

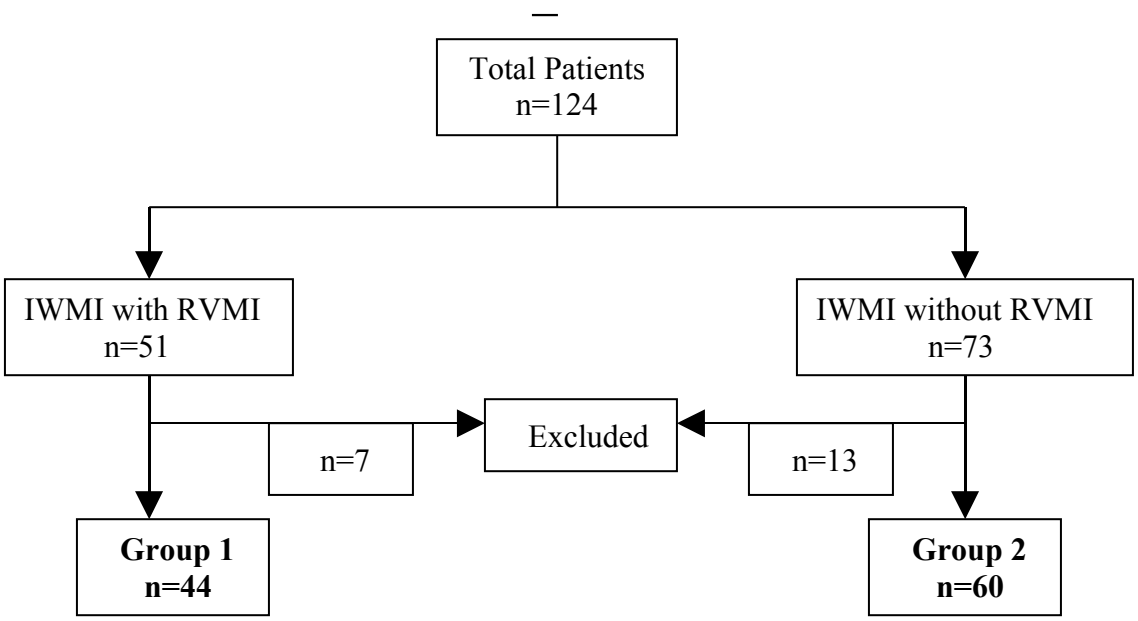
Group 2 – Patients with IWMI without RVMI (n = 73)

Out of the 104 patients, 20 patients (7 from Group 1 & 13 from Group 2) were excluded from the study due to various reasons described below.

20 patients were excluded from the study due to:

- Delayed presentation >2 days after onset of chest pain (n=8)
- Presence of preexisting cardiac dysfunction due to myocardial infarction, cor pulmonale, DCM or valvular heart disease (n=6)
- Atrial fibrillation (n=1)
- Technically inadequate echo (n=5).

Finally, Group 1 (IWMI with RVMI) consisted of 44 patients and Group 2 (IWMI without RVMI) consisted of 60 patients, and these 104 patients underwent clinical evaluation, investigations including EKG, Echocardiography and coronary angiography in whom-ever it was feasible.



Detailed history was obtained from all the patients, including the presence of risk factors like

-diabetes mellitus,

-hypertension,

-smoking and

-family history of ischemic heart disease.

Baseline investigations were done in all patients including complete blood count, blood sugar, renal function tests, lipid profile, chest X-ray. Cardiac enzymes, namely, transaminase levels, Creatinine kinase and CK-MB were done in all patients.

Patient Characteristics:

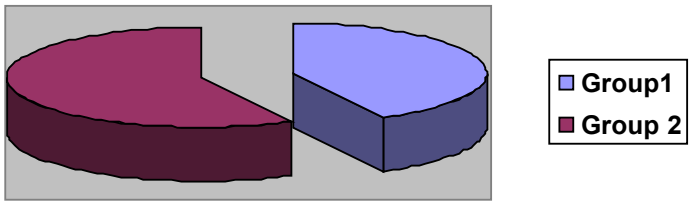
Table 2. Patient's characteristics

	Group 1 (n=44)	Group 2 (n=60)	TOTAL
Age <40 Yrs	6	7	104
40-60 yrs	28	40	
>60 yrs	10	13	
Male	34	44	78 (75%)
Female	10	16	26 (25%)
Hypertension	18	22	40 (38%)
Diabetes	26	22	48 (46%)
Dyslipedemia	17	13	30 (29%
Smoking	32	28	60 (58%)
F/H of CAD	13	11	24 (23%)

Patient's clinical baseline profile:

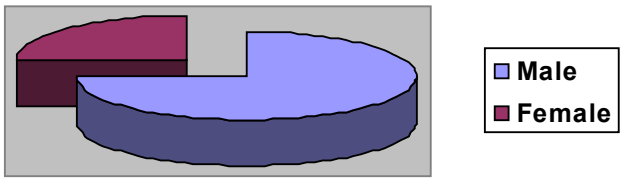
Clinical parameters	Group 1 (n=44)	Group 2 (n=60)
Average age (yrs)	54 ± 11.3	51 ± 10.5
Systolic BP (mmHg)	108 ± 14	114 ± 12
Diastolic BP (mmHg)	74 ± 8	76 ± 10
Heart rate (per min)	70.1±7.4	68.3 ± 8.4
Thrombolysis with SK	30	42
CK-MB values	115 ± 40	75 ± 26

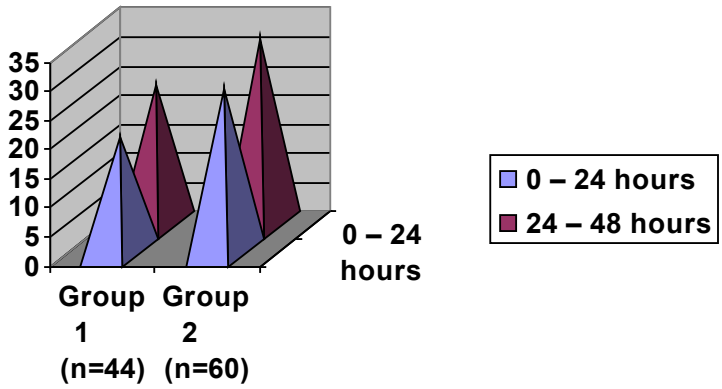
Table 3. Patient's baseline clinical profile



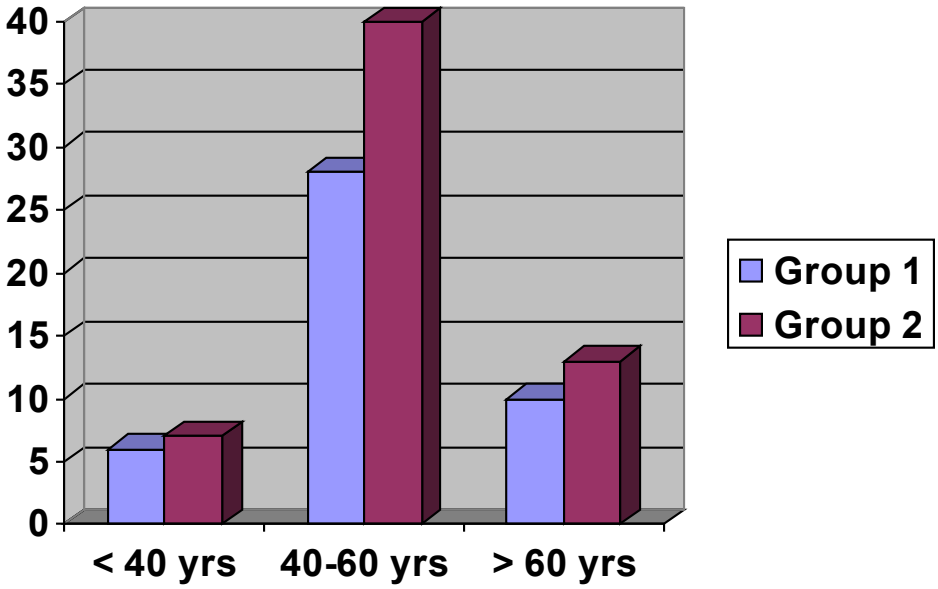
Group 1 = Patients with RVMI
Group 2 = Patients without RVMI

Sex distribution

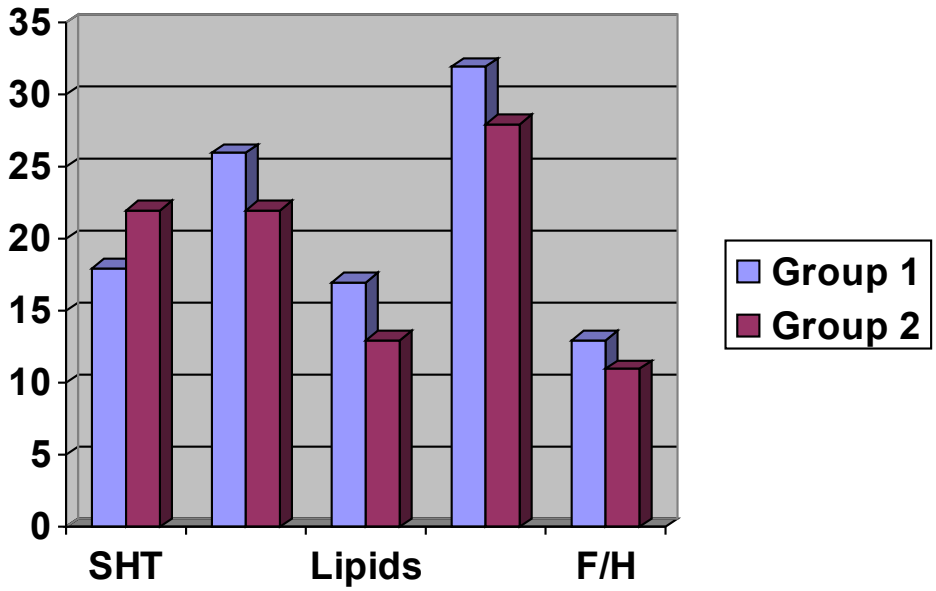




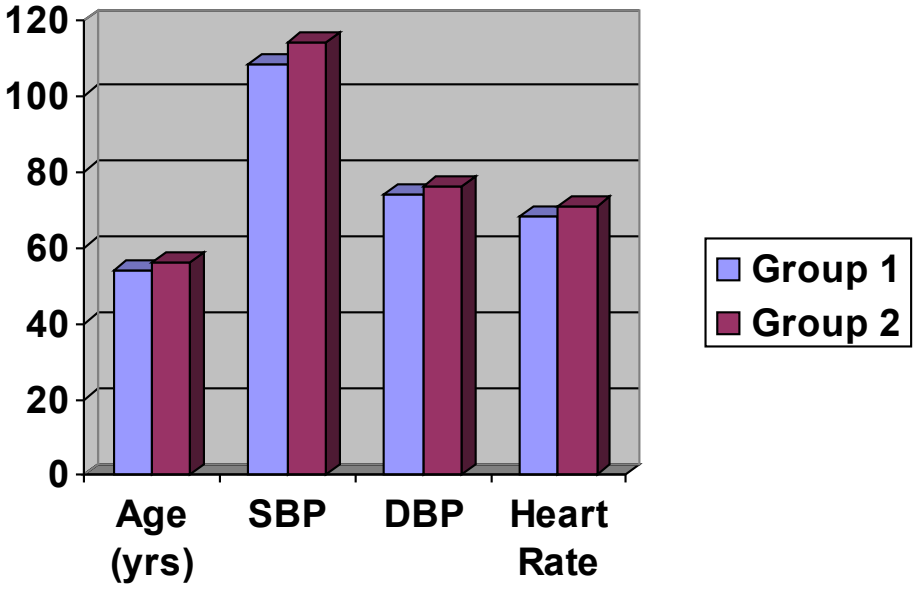
Time to echo



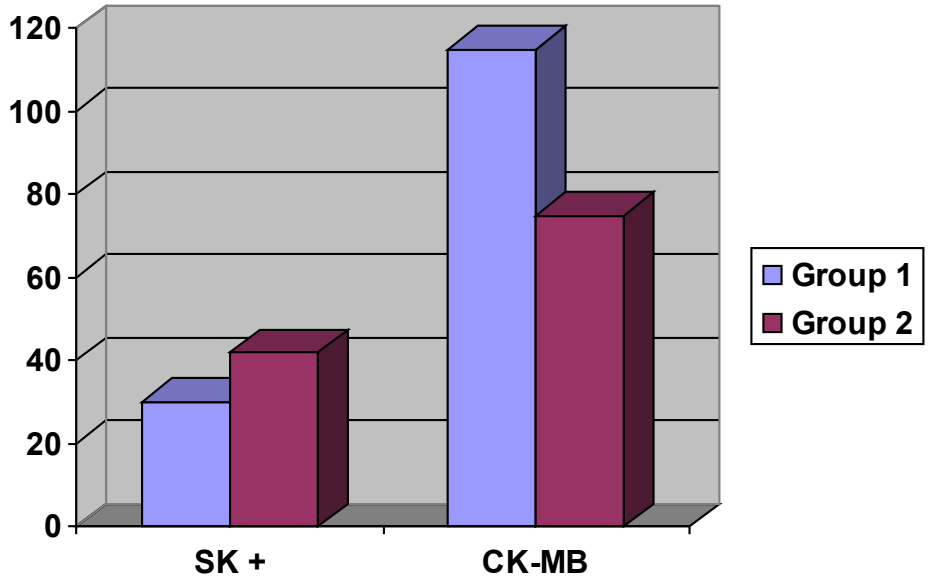
Age - wise distribution



Risk factors



Baseline profile of patients



Patients who were in the time window for thrombolysis, and who had no absolute contra-indications for thrombolysis were given Inj. Streptokinase I.V over 45 minutes via an infusion. All patients were treated with antiplatelets, statins, and intravenous fluid replacements whenever needed. Those who developed significant atrio-ventricular blocks were managed with temporary pacemaker implantation. Those in shock were managed with fluid replacement and inotropic support. All patients were closely monitored during the intensive period.

Echocardiography was done in all patients within 48 hours of symptom onset, within 24 hours whenever feasible. All patients were advised coronary angiography, and CAG was done ultimately in 74 patients within 30 days of the acute coronary unit. CAG was done through the femoral route, using properly sized sheath, and Judkin's catheter, Amplatz when necessary.

Time to echo	**Group 1 (n=44)**	**Group 2 (n=60)**	**Total**
0 – 24 hours	20	28	48
24 – 48 hours	24	32	56
Total	44	60	**104**

Table 4. Time interval between symptom onset and echocardiography

Coronary angiography was not done in 38 patients because:

- Patients not willing for the same (n=24)
- Elevated renal parameters (n=6)
- Died due to complications of MI (n=8)

Definition of Inferior wall myocardial infarction and right ventricular myocardial infarction:

History & Enzymes:

Acute myocardial infarction was diagnosed by the presence of typical chest pain lasting more than 30 min and an increase of greater than two times normal values in aspartate transaminase (30 U/liter) and CPK (170 U/liter) activity within 24 h after admission.

Electrocardiography:

ECG changes, including an ST segmental elevation of greater than 0.1 mV with or without Q waves in at least two of the leads II, III, and aVF, also were required. In all patients, a standard 12 lead ECG and right precordial leads (V3 to V6R) were recorded at hospitalization. A 1 mm ST segment elevation with or without Q waves in V4R lead was required to diagnose the involvement of the right ventricle[32].

Echocardiography:

Transthoracic echocardiography was performed in each patient using a Philips iE 33 echocardiography machine with a 3.5 MHz transducer including second harmonic and tissue Doppler imaging technology. Echocardiography was done within 24 hours of symptom onset whenever technically possible and in all patients within 48 hours of symptom onset. Subjects were examined in supine, left-lateral position. They underwent conventional M-mode and two-dimensional echocardiography from a left parasternal and apical window. The baseline LV volumes and ejection fraction were calculated by Simpson's biplane method.

The following measurements were done using Philips IE 33. RT echo machine:

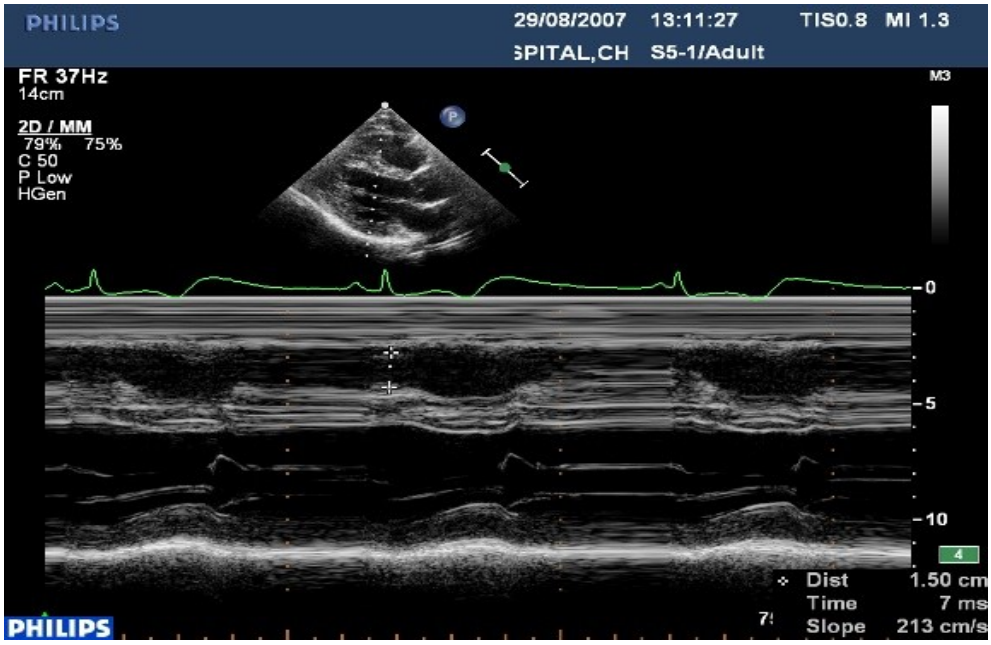
- RV dimensions
- RV contractile function & Inter-ventricular septum movement
- Tricuspid regurgitation jet & Pulmonary artery systolic pressure
- Tricuspid annular movement - M-mode
- Doppler tissue imaging – lateral & septal wall systolic / early, late diastolic velocities
- Myocardial performance index (Tei index)

RV dimensions:

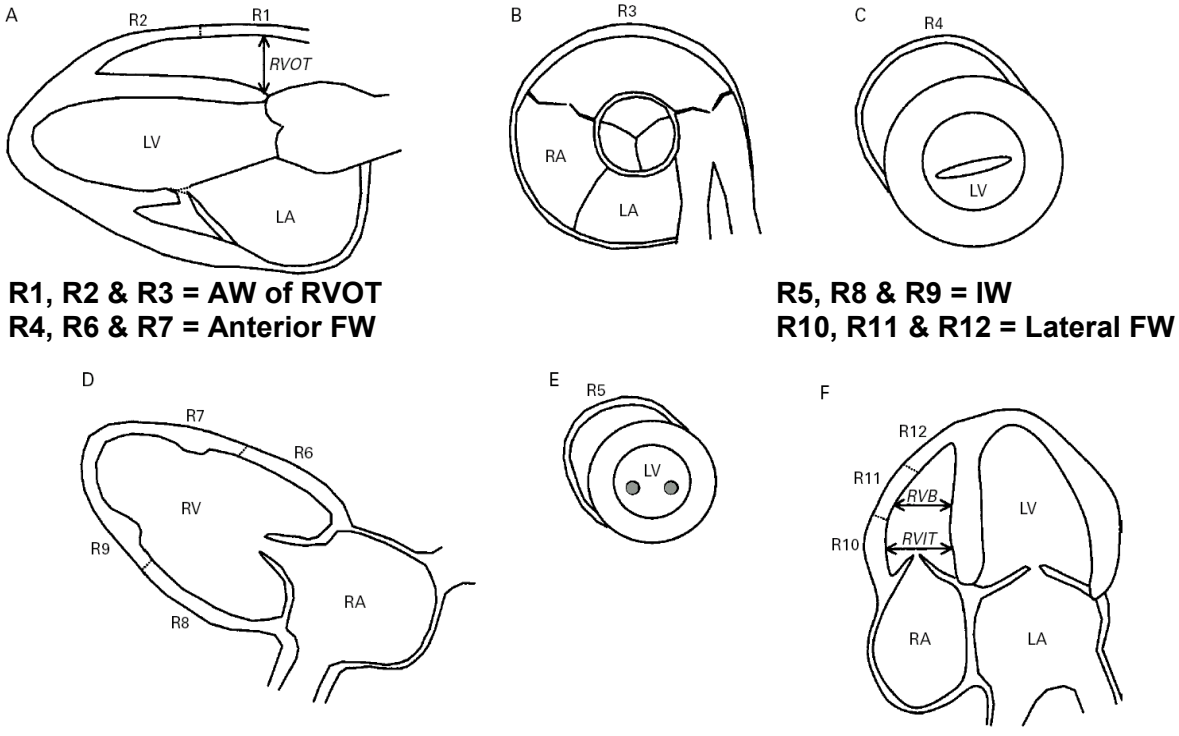
Right and left ventricular end diastolic diameters in the supine position using the same M-mode cross-section in the parasternal long axis view. Normal values were defined as less than 26 mm for the right ventricular end diastolic diameter, greater than 37 mm for the left ventricular end diastolic diameter, and less than 0.5 for the ratio of the right ventricular diameter to the left ventricular diameter[26].

Right ventricular contractile function:

Right ventricular wall motion was assessed in parasternal long axis, parasternal short axis, RV inflow and apical four chamber views. The right ventricle was divided into anterior wall of RVOT, anterior free wall, lateral free wall and inferior wall. Each segment was analyzed as normal, hypokinetic, akinetic and dyskinetic.



Picture 2. The measurement of RV-EDD by M-mode in PLAX



Picture 3. Segments of right ventricle used to measure contractile dysfunction

IVS motion:

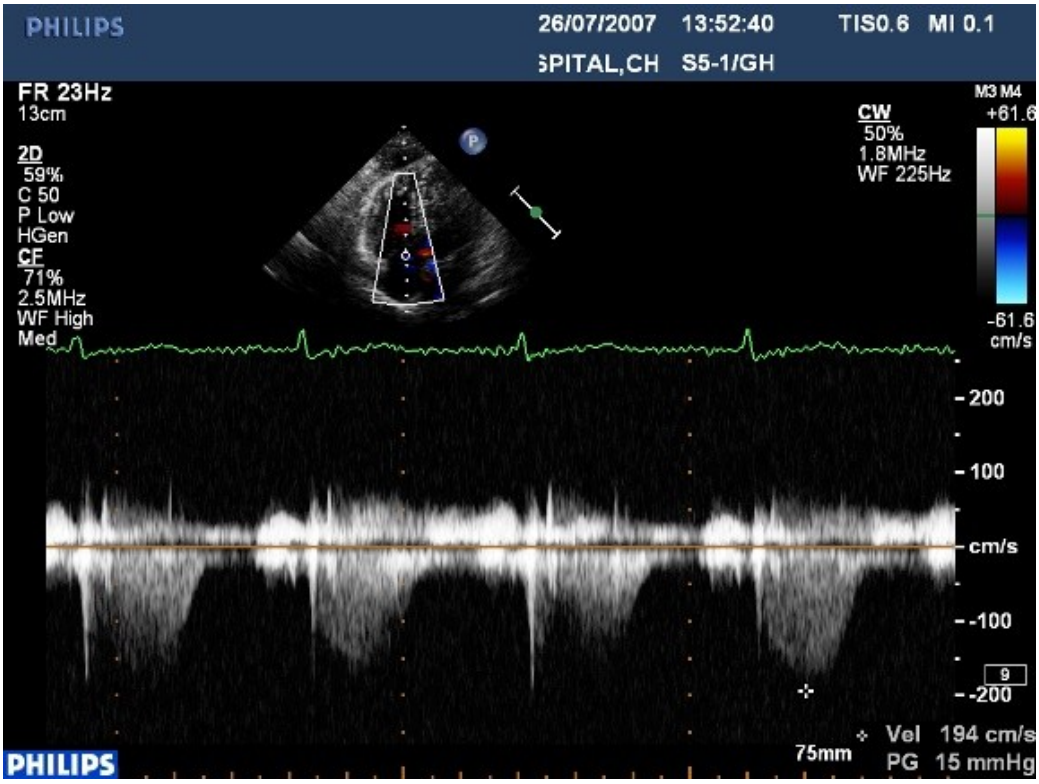
Interventricular septal motion was analyzed from the parasternal long axis and apical four chamber views, and it was considered abnormal if an anterior systolic motion of the septum occurred[26].

Tricuspid regurgitation jet:

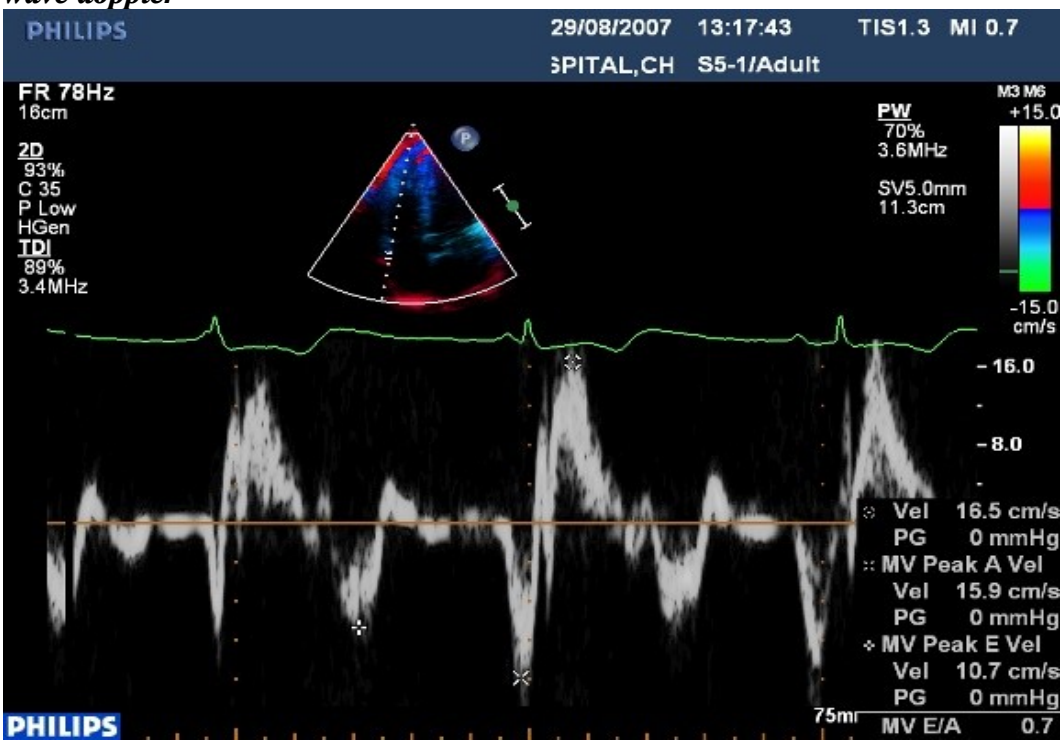
Tricuspid regurgitant flow peak velocity was recorded from parasternal short axis and apical four chamber views using colour doppler and scored as 0-none, 1-mild, 2-moderate and 3-severe TR. The systolic tricuspid regurgitation pressure gradient between the RV and the right atrium was calculated by the simplified Bernoulli equation.

Tissue Doppler Index:

Pulsed wave TDI of the systolic tricuspid annular motion (cm/s) at the lateral free wall (TVlat) and at the septal wall (TVsept) was obtained from the apical 4-chamber view using a pulsed wave doppler sampling gate of 2–4 mm and a sweep of 100–150 mm/s. A major positive Sm was recorded with the movement of the annulus toward the cardiac apex during systole. Two major negative velocities were recorded with the movement of the annulus toward the base of the heart during diastole, as follows: one during the early phase of diastolic myocardial velocity (Em) and another during the late phase of diastolic myocardial velocity (Am). The acoustic power and filter frequencies were adjusted for detecting myocardial velocities. The average of 3 TDI signals from different cardiac cycles was employed for data analysis.



Picture 4: Measurement of tricuspid regurgitation jet and PASP measurement using continuous wave doppler

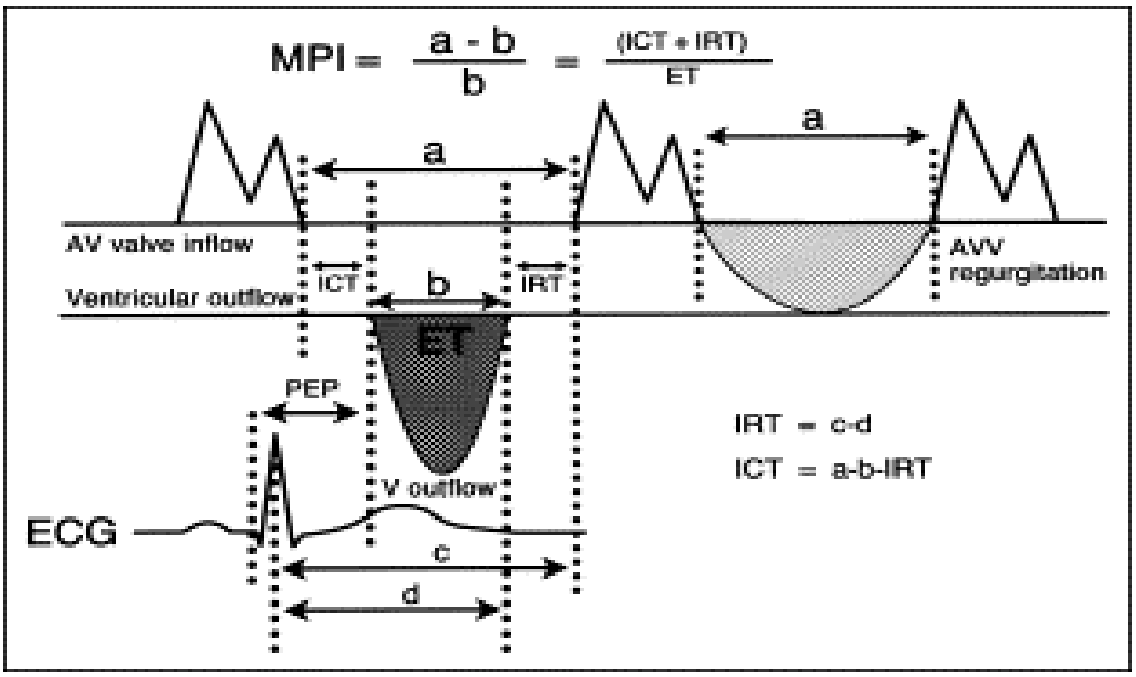


Picture 5. Tissue doppler of RV myocardium with sample volume in the RV lateral wall showing Sm, Em & Am tissue velocities.

Myocardial Performance Index:

Right ventricular myocardial performance was calculated as follows. The tricuspid inflow is interrogated by pulse wave doppler in the apical 4 chamber view and the time interval from end of ‘A’ wave to the onset of the following ‘E’ wave is noted as ‘a’ in milliseconds. Then the pulmonary valve is interrogated by pulse wave doppler in the parasternal short axis view. The total pulmonary ejection time is noted as ‘b’. 5 consecutive cardiac cycles were averaged to obtain each value for ‘a’ and ‘b’ to correct for heart rate variation and measurement errors.

The time interval ‘a’ denotes combination of isovolumetric contraction time (IVCT), ejection time (ET) and isovolumetric relaxation time (IVRT). As the doppler period ‘b’ is the pulmonary ET, the sum of IVCT and IVRT is derived by subtracting ‘b’ from ‘a’. MPI is calculated as MPI = a-b/b and a valueless ratio is obtained.



Picture 6. Measurement of right ventricular MPI

Coronary angiography:

Coronary angiography was done in 66 patients out of 104. All of them underwent coronary angiography within one month of the myocardial infarction. 24 patients were not willing for coronary angiography. 6 patients had elevated renal parameters and hence CAG was not performed. 8 patients had died due to complications before coronary angiography could be planned. The coronary angiography was performed via the femoral artery route using standard sized sheaths and Judkin's left and right coronary catheters, Amplatz if necessary. Multiple angulations and views were used. Lumen diameter narrowing was graded as 0, 25, 50, 75, 90 and 100%. The definition of a significant anatomical stenosis was >70% localized luminal narrowing.

The coronary angiography was considered indicative of an RVMI if an occlusion of the right coronary artery was present proximal to the acute marginal branches[31] or the major right ventricular branches.

Studies have shown than a direct correlation exists between the anatomic site of right coronary artery occlusion and the extent of right ventricular infarction. If occlusion occurs before the right

ventricular marginal branches, and collateral blood flow from the left anterior descending coronary artery is absent, then the size of infarction generally is greater.

In a similar study by Dokainish et al[24], patients with inferior wall myocardial infarction underwent coronary angiography and the presence of RVMI was determined by the presence of the infarct culprit lesion (ICL) proximal to the first RV branch of the right coronary artery (RCA).

Complications:

Complications occurring during the in-hospital period were determined for all patients. The major in-hospital complications evaluated were hypotension (systolic blood pressure less than 100 mm Hg), cardiogenic shock (systolic blood pressure less than

Statistical analysis:

Data are presented as mean ± SD for descriptive statistics. We chose the average values for 3-time measurements. Continuous data are expressed as mean ± 1 SD. Comparison of measurements between patients with inferior myocardial infarction with right ventricular infarction and patients without right ventricular involvement was performed using a two tailed Student's *t*-test. A probability (*P*) value of less than 0.05 was considered significant.

.

RESULTS

During the study period, 104 patients were evaluated and underwent a complete echo-Doppler examination. Among these patients, 44 had an RVMI, and constituted Group 1, and the remaining 60 patients had an inferior wall myocardial infarction without right ventricular involvement (Group 2).

Most of the patients were in the age group of 40-60 years (68/104 = 65%).

Most of the study population were male patients, with 78 out of the104 patients being male (75%), the rest 26 patients were female.

As far as the history was concerned, 40/104 (38%) were hypertensives, 48/104 (46%) had a history of diabetes mellitus, 60/104 (58%) of patients were smokers, and a family history of ischemic heart disease was evaluated in 24/104 patients (23%).

Various complications were noted in the study patients, the commonest being conduction disturbances.

- Conduction disturbances were noted in 15/44 patients (34%) of Group 1, and in only 6/60 (10%) of patients in Group 2.
- Cardiogenic shock complicated 3/44 patients (7%) of Group 1, and none of the patients in Group 2.
- Pulmonary artery systolic pressures >30mmHg were noted in 30/44 (69%) of Group 1, while in Group 2 patients, only 1/60 patients (2%) had PASP exceeding 30mmHg.

8 patients died due to complications – of which 7 patients belonged to Group 1 and 1 patient belonging to Group 2 died. All patients died within 1 week of the acute event. Elevated pulmonary

artery pressures were noted in 7/8 patients who died with a mean pulmonary artery systolic pressures of 40mmHg. We can hypothesize that patients with persisting sign of pulmonary hypertension did not experience reperfusion of the related artery and had an unfavorable course[30].

Electrocardiography:

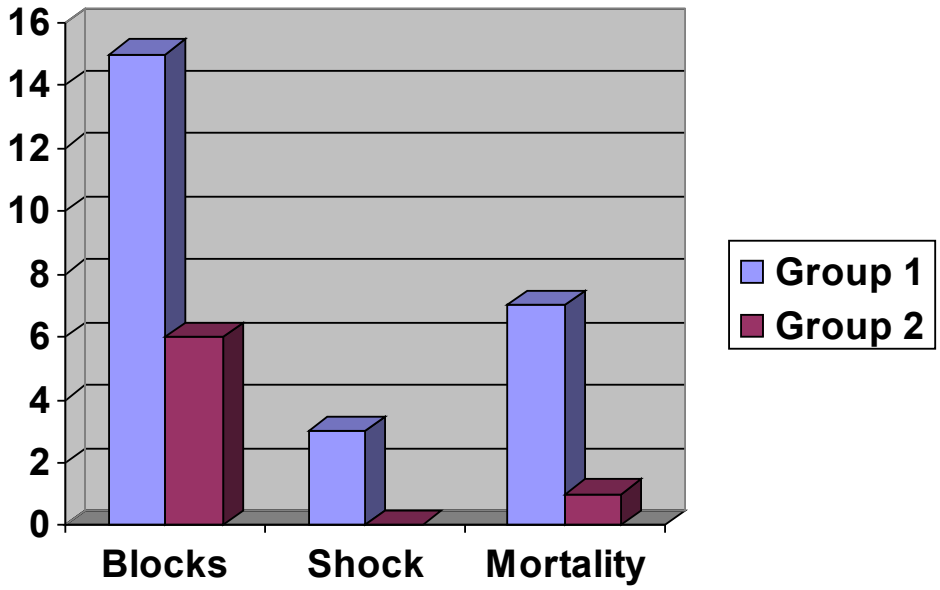
12 lead electrocardiography was done in all patients. Right sided chest leads and posterior wall leads were also placed and ECG recorded in all patients. By using the criteria described in methods, the culprit artery was identified as

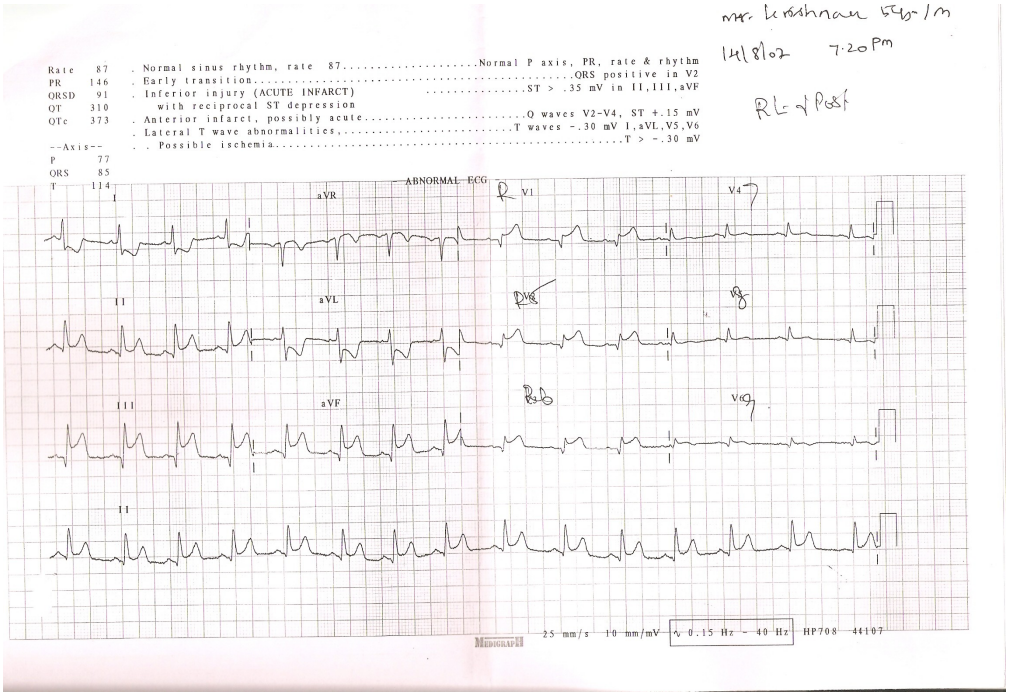
- Proximal RCA – 42 patients
- Distal RCA – 45 patients
- Left circumflex – 17 patients

Evidence of right ventricular involvement as defined by 1mm ST elevation in V4R was fund in 38 patients (of which, 34 patients had proximal RCA as the culprit vessel on ECG and 4 had left circumflex as the vessel causing myocardial infarction).

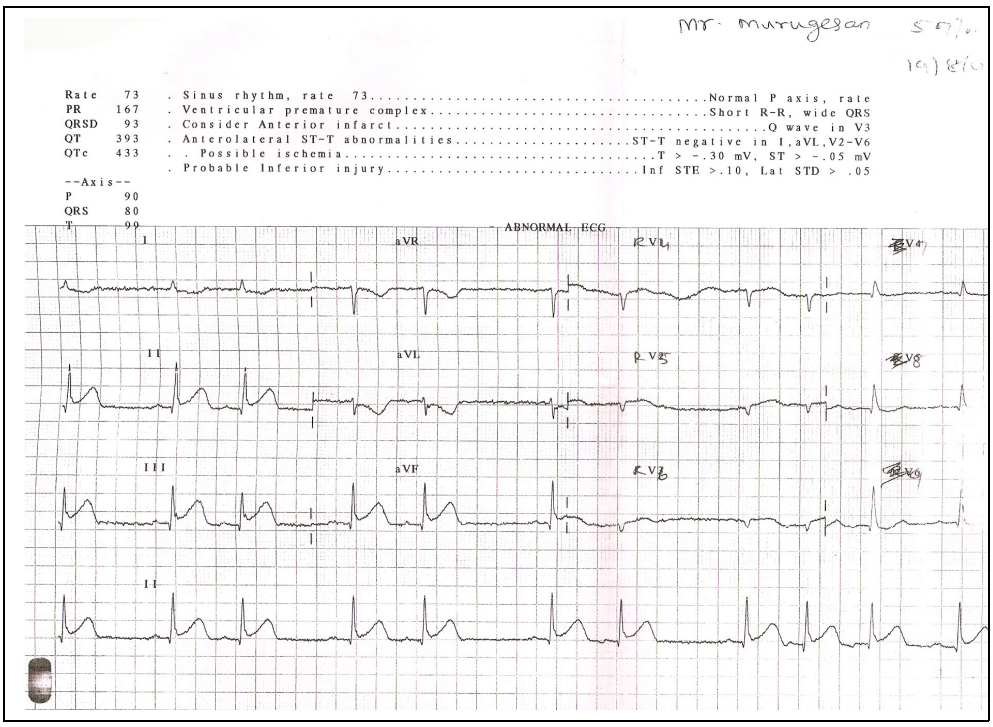
	Group 1 (n=44)		Group 2 (n=60)		Total
	SK+ (n=30)	SK–(n=14)	SK+ (n=42)	SK–(n=18)	
SA/AV blocks	10 (33%)	5 (36%)	4 (9%)	2 (11%)	21 (20%)
Shock	1	2	-	-	3 (3%)
PASP >30	20 (68%)	10 (70%)	1 (2%)	-	16 (15%)
Mortality	5 (16%)	2 (14%)	-	1 (6%)	8 (7%)

Table 5. Incidence of complications in the study population





Picture 7. ECG of a patient with proximal RCA occlusion: IWMI with RVMI



Picture 8. ECG of a patient with distal RCA occlusion: Distal RCA occlusion

Echocardiography:

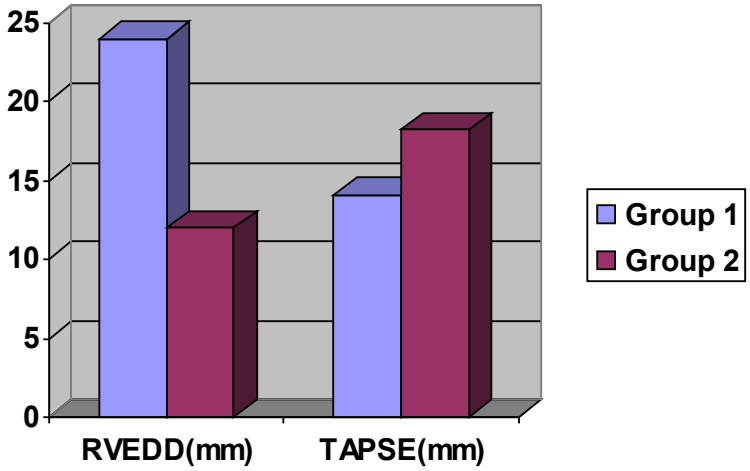
Echocardiography was done in all 104 patients within 48 hours after the onset of the acute event, of which, echocardiography was done within 24 hours of symptom onset in 48 patients. The results of the investigation is as below:

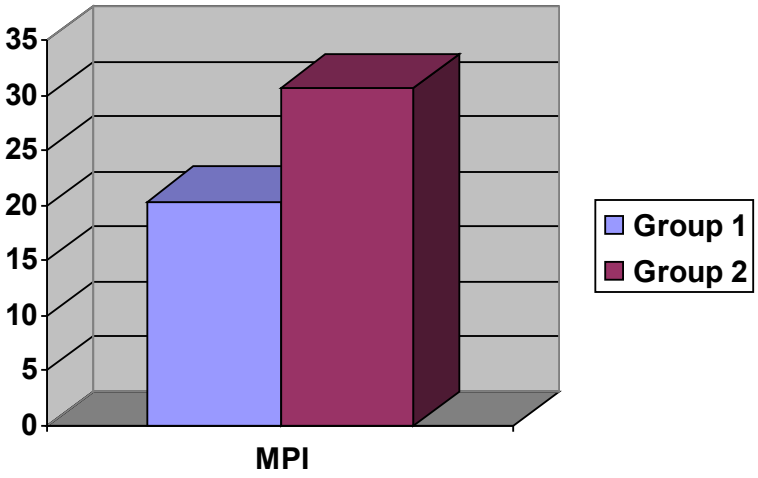
- RV end-diastolic diameter was increased in patients with RVMI, compared with those without RV involvement (Picture 9), although it failed to achieve statistical significance (p=0.1).
- RV contraction abnormalities and IV septal motion abnormalities were noted in 41% of patients with RVMI, and only in 3% of patients without it. Significantly, the wall motion abnormalities were noted in those patients who had their echo done within 24 hours of symptom onset, stressing the fact that, the right ventricle recovers fast after an ischemic insult.
- Tricuspid regurgitation was noted in nearly half of the patients with right ventricular involvement; this regurgitation, when quantified by colour doppler, was mild in a majority of cases, moderate in some and severe in none.
- The pulmonary artery systolic pressure was slightly elevated and above normal in patients of Group 1, compared to Group 2 (Picture 10), statistically not significant (p=0.2)
- The tricuspid annular excursion was statistically significantly decreased in patients with right ventricular myocardial infarction, signifying that RV systolic function was depressed in those patients (p=0.05) (Picture 11 & 12).
- Myocardial performance index was found to be statistically significant, it is raised to nearly two fold the reference values in patients with RVMI; the values were not much high compared to controls in those without right ventricular involvement (Pictures 13 – 16). (p=0.01).
- Tissue doppler was done in the right ventricular free wall and the septal regions and the systolic (Sm), early diastolic (Em) and late diastolic (Am) velocities were noted. It was found that the right ventricular free wall Sm and Em values were statistically significantly depressed in patients with right ventricular myocardial infarction (p=0.02). The values were near normal in those without RV involvement. However, the RV free wall late diastolic velocities (Am) and

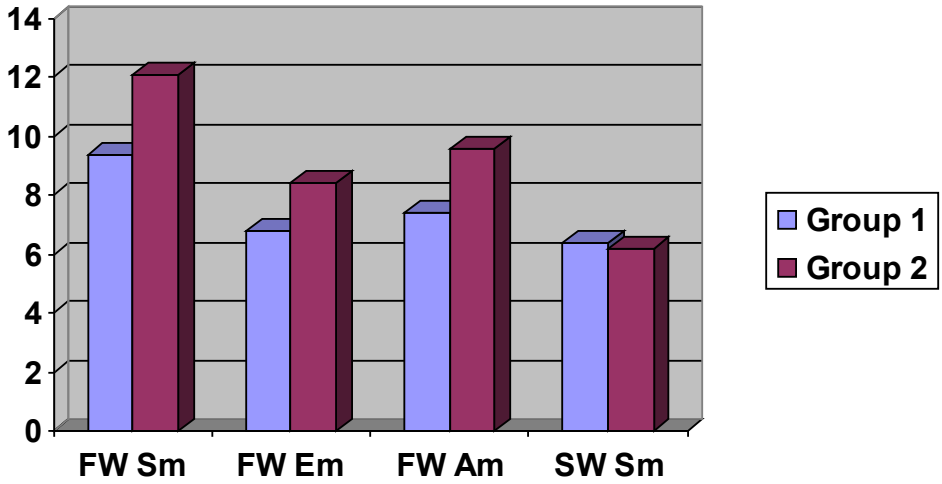
the septal velocities did not differ much in patients with and without right ventricular myocardial infarction (Pictures 17,18). (p=0.22)

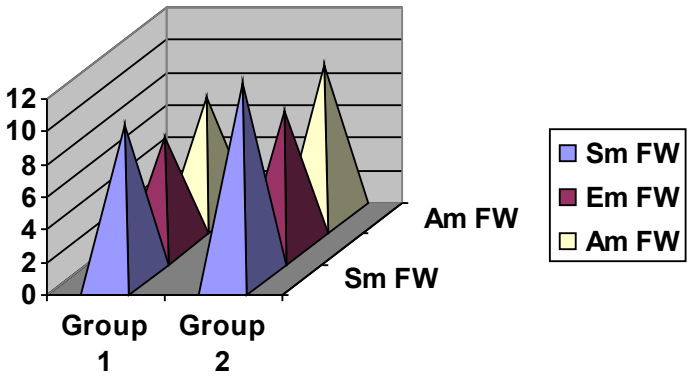
Parameters	**Group 1 (n=44)**	**Group 2 (n=60)**
RV EDD (mm)	21.8 ± 4.1	11.3 ± 3
RV contraction abnormalities	18/44 (41%)	2/60 (3%)
IVS motion abnormality	15/44 (31%)	1/60 (2%)
Tricuspid regurgitation	21/44 (48%)	6/60 (10%)
PASP (mmHg)	31.8±2.7	17.1 ± 2.8
TAPSE (mm)	14.1 ± 1.2	18.2 ±0.71
MPI	0.44 ± 0.07	0.28 ± 0.02
TDI – RV Free wall Sm	9.45 ± 0.49	12.1 ± 0.52
Em	6.8 ± 0.34	8.3 ± 0.65
(cm/sec) Am	7.4 ± 0.67	9.4 ± 0.65
TDI – Septal wall (cm/sec) Sm	6.3 ± 0.5	6.2 ± 0.5

Table 6. Values of various echocardiographic parameters in patients presenting with IWMI with or without RVMI


25
20
15
10
5
0
RVEDD(mm)
TAPSE(mm)
Group 1
Group 2


35
30
25
20
15
10
5
0
MPI
Group 1
Group 2


14
12
10
8
6
4
2
0
FW Sm
FW Em
FW Am
SW Sm
Group 1
Group 2



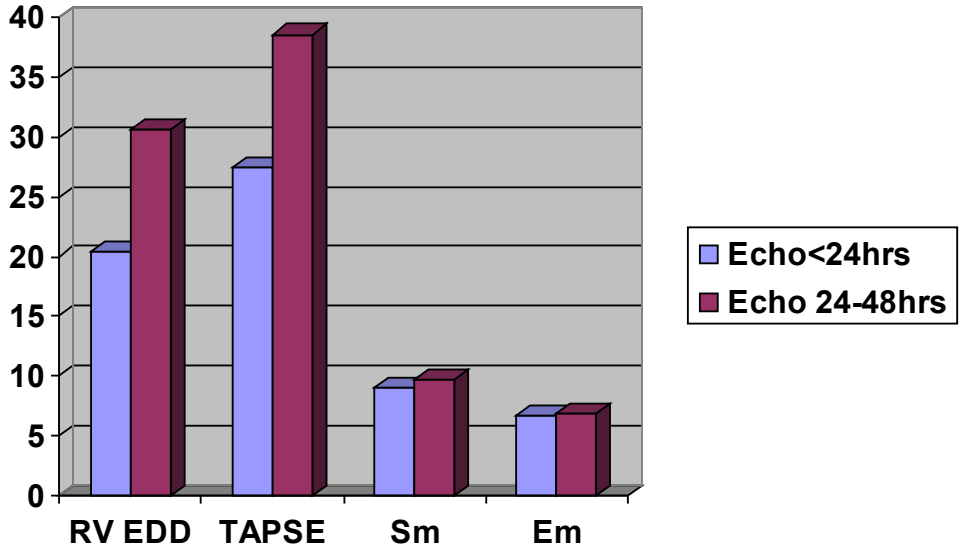
Sub group analysis:

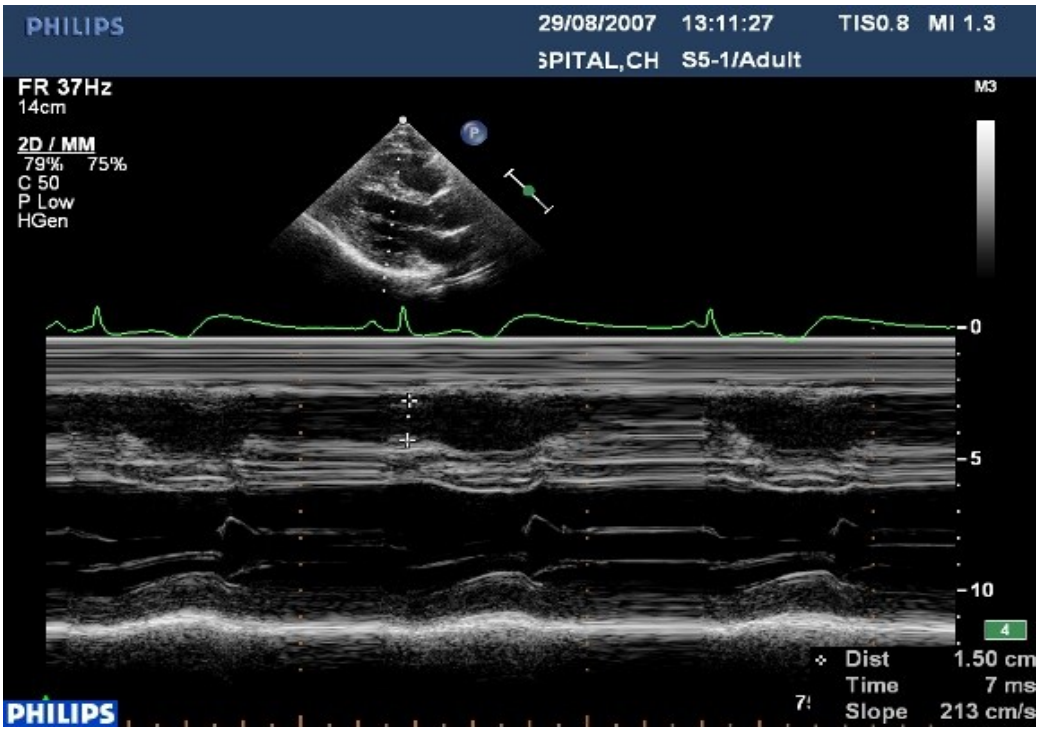
- All echocardiographic parameters did not statistically significantly differ in the various age groups (p=0.5).
- Similarly, the parameters also did not statistically significantly differ in both sexes, revealing that RV dysfunction was present equally in both males and females.
- As far as thrombolysis is concerned, RVEDD was higher, TAPSE was lower, Sm & Em values were lower in patients thrombolysed when compared to those who were not; but, they were statistically insignificant (p=0.2). Only, myocardial performance indeed was statistically significantly low (p=0.04) in patients who were thrombolysed with streptokinase when compared to those who were not.
- Echocardiography done within 24 hours of the acute event, showed significantly lower Sm scores and very significant high MPI scores in patients with right ventricular myocardial infarction. Although RVEDD and PASP were high and TAPSE was low when echo was done within 24 hours, compared to those done between 24-48 hours, it lacked statistical significance (p=0.1).

Echocardiographic sub group analysis revealed the following values:

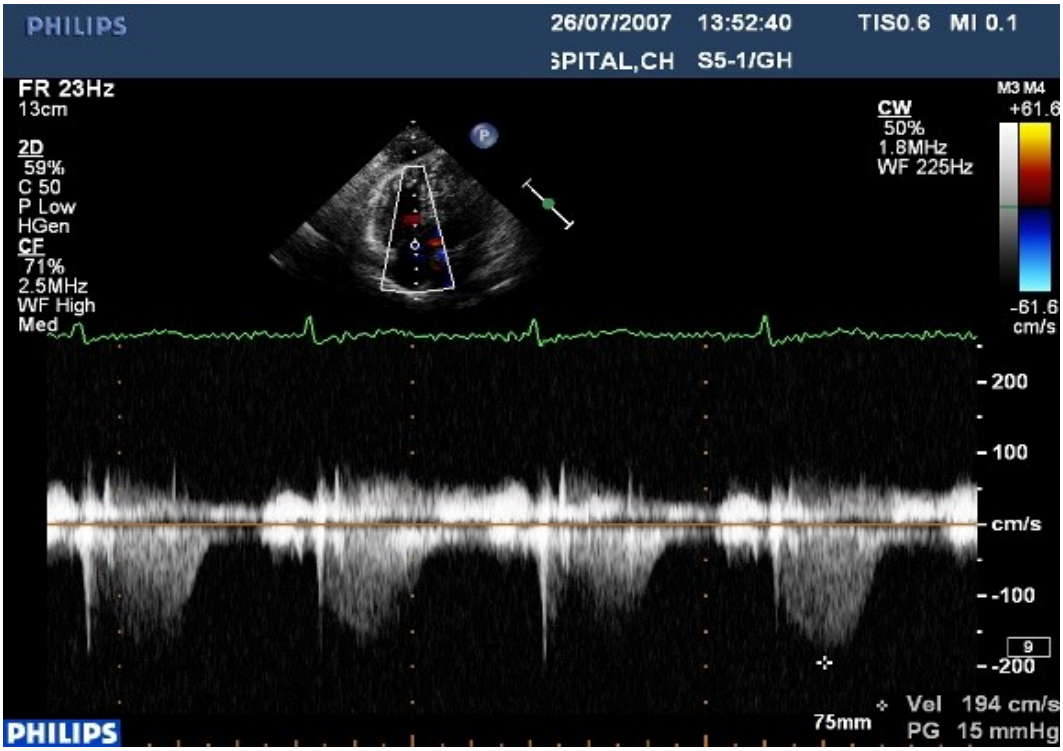
Sub groups In RVMI	**RV EDD (mm)**	**TAPSE (mm)**	**PASP (mmHg)**	**Sm (cm/sec)**	**Em (cm/sec)**	**MPI**
Age <40yrs	14.2 ±	14.4 ±.7	18 ± 4	9.5 ± 0.7	6.7 ± 0.6	0.31 ± .
40-60 yrs	4.1					05
	25.1 ±	16.4 ±.9	28 ± 8	9.6 ± 0..6	6.7 ± 0.5	0.34 ± .
60 yrs	5.2					06
	28 ± 2.1	17.2	36 ± 4	9.3 ± 0.2	6.5 ± 0.1	0.38 ± .
		± .9				02
Sex - Male	22.2 ±	17.2	24 ± 6	9.5 ± 0.6	6.7 ± 0.6	0.32 ± .
Female	4.3	± .8				05
	21.1 ±	16.8	22 ± 4	9.3 ± 0.4	6.7 ± 0.3	0.34 ± .
	5.1	± .2				01
SK - given	18.3 ±	16.8	21 ± 5	9.6 ± 0.6	6.8 ± 0.4	0.36 ± .
Not given	2.2	± .4				04
	22.1 ±	14.1	31 ± 8	9.3 ± 0.7	6.7 ± 0.4	0.49 ± .
	3.2	± .3				09
Echo <24hrs	27.3 ±	14 ± 1.2	34 ± 6	9.0 ± 0.3	6.7 ± 0.3	0.51 ± .
24-48 hrs	5.4					08
	11.1 ±	17.2	18 ± 7	9.7 ± 0.5	6.9 ± 0.6	0.39 ± .1
	4.5	± .9				

Table 7. Subgroup analysis of echocardiographic parameters in study patients

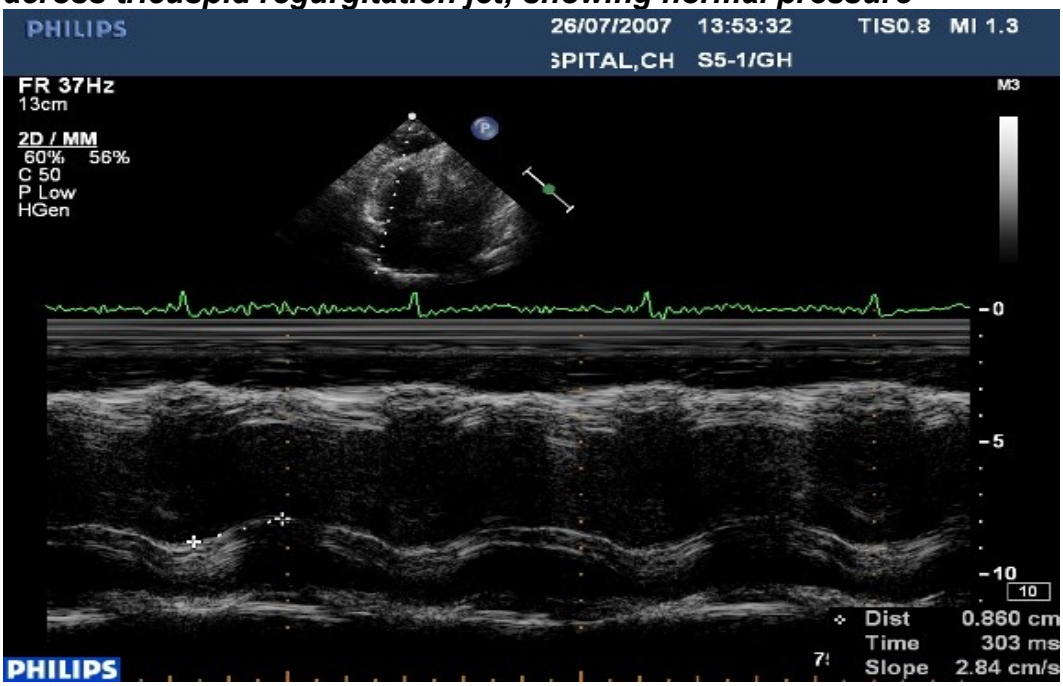




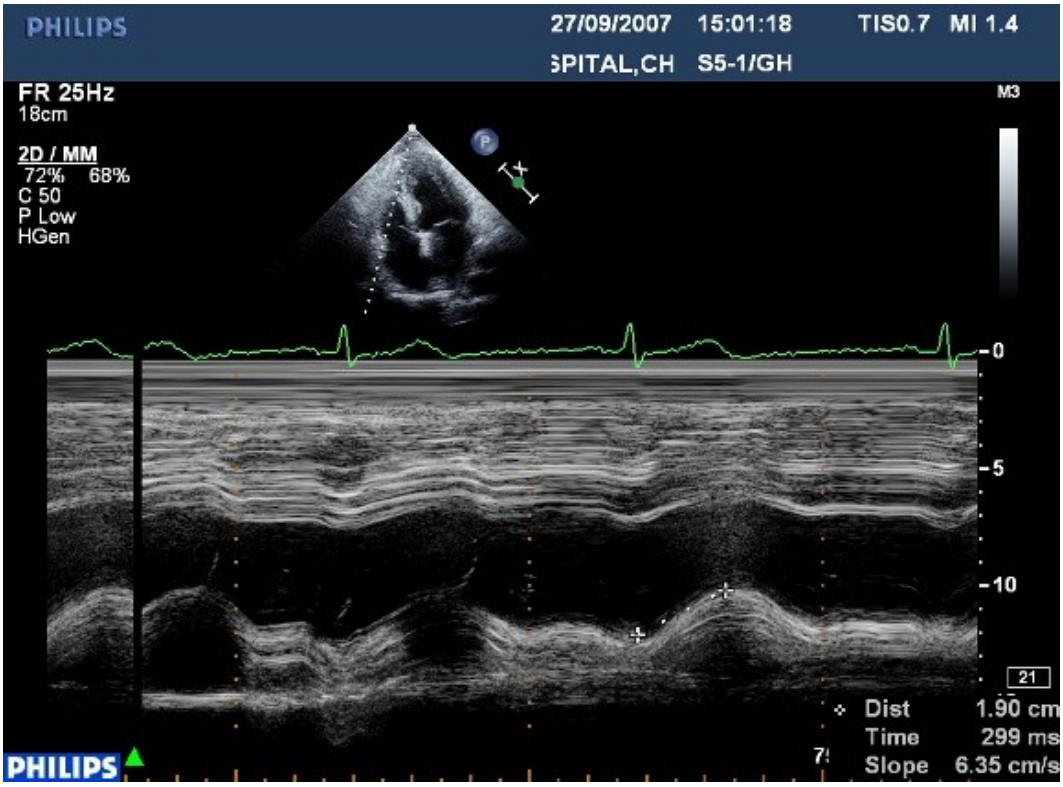
Picture 9. Measurement of RV internal diameter by M-mode in PLAX. Normal value in a patient of IWMI without RVMI



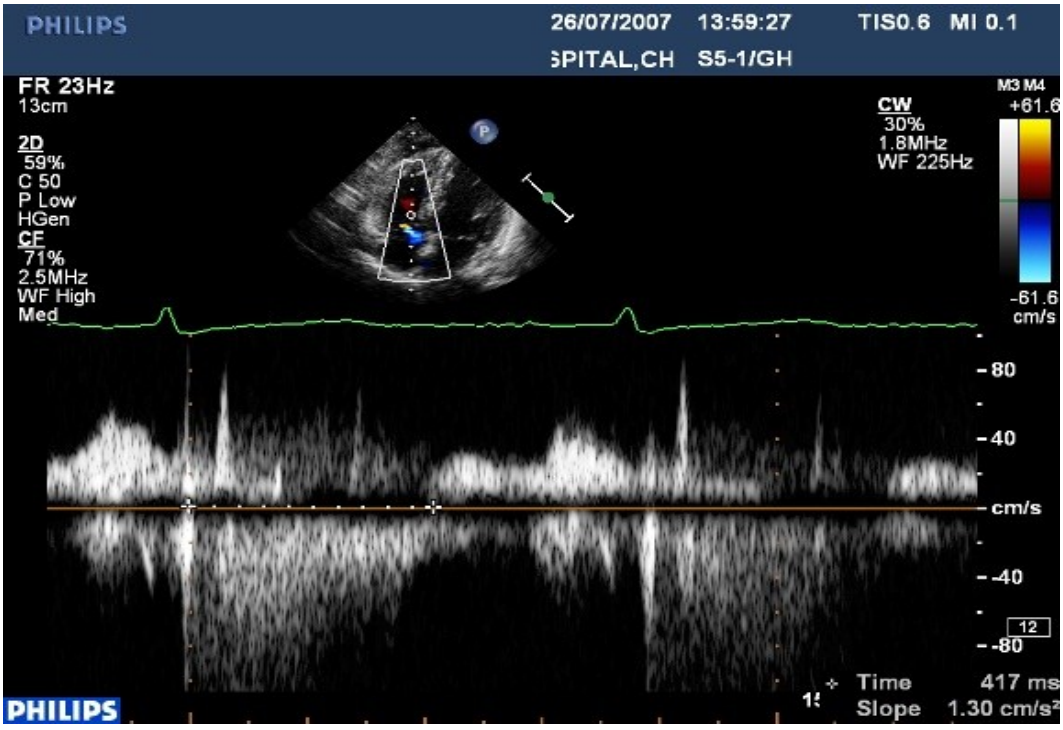
Picture 10. Measurement of RV systolic pressure using continuous wave doppler across tricuspid regurgitation jet; showing normal pressure



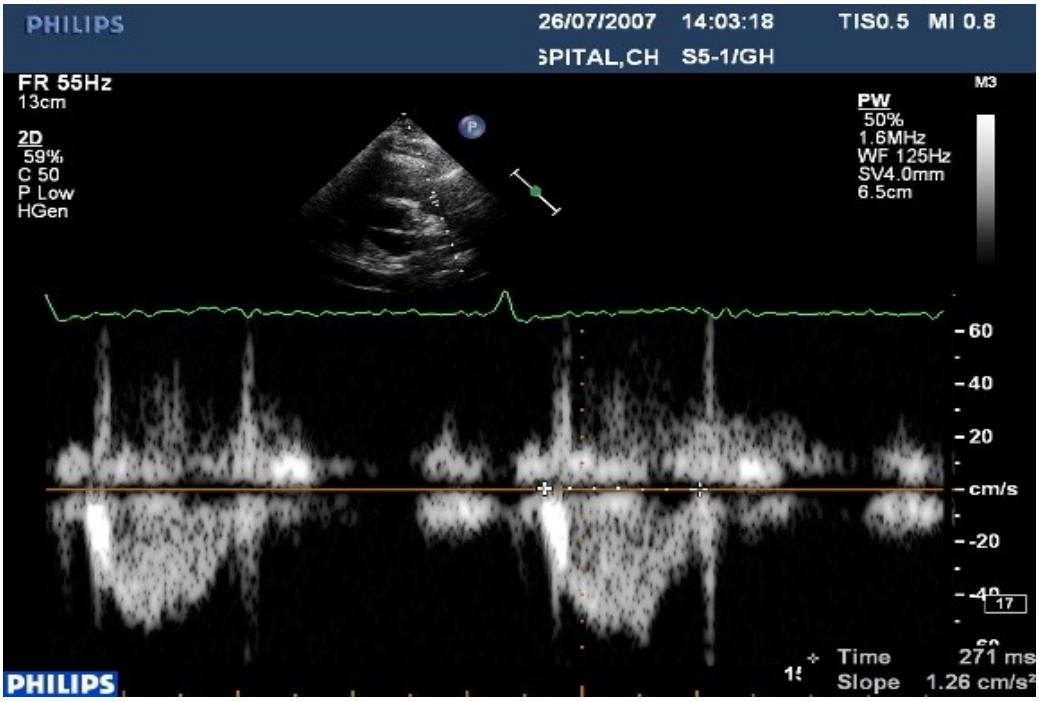
Picture 11. Measurement of Tricuspid annular movement by M-mode in apical 4 chamber view; reduced excursion in a patient of IWMI with associated RVMI.



Picture 12. Measurement of Tricuspid annular movement by M-mode in apical 4 chamber view; normal excursion in a patient of IWMI without RVMI.

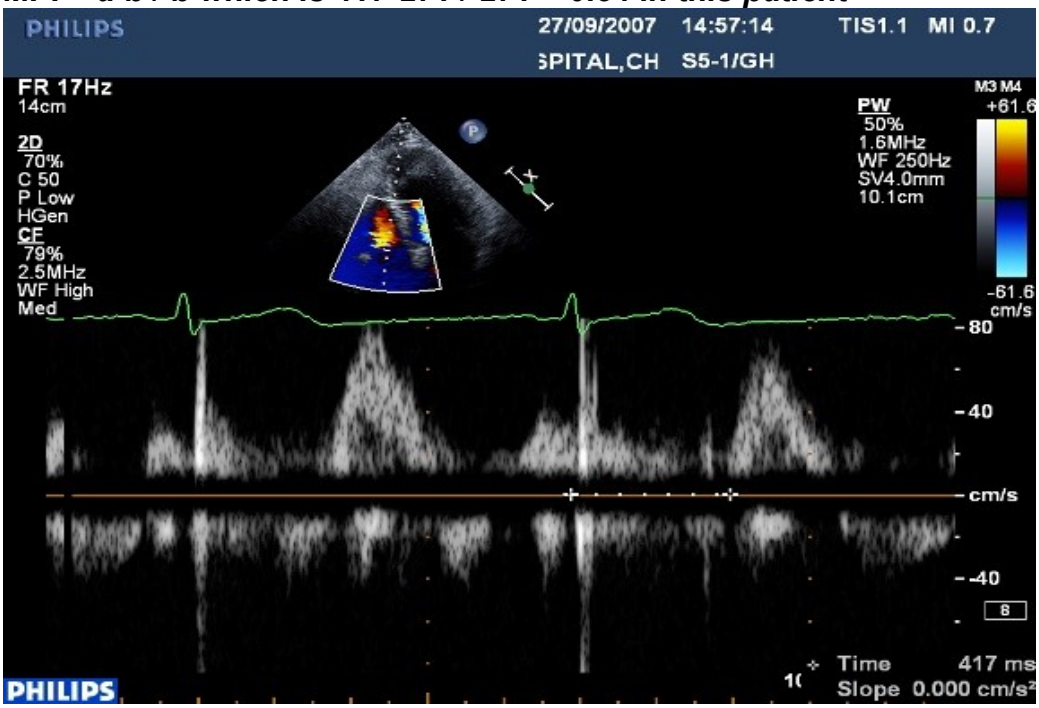


Picture 13. Measurement of MPI: Value 'a' which is IVCT + ET + IVRT in apical 4 chamber view- a patient of IWMI with RVMI

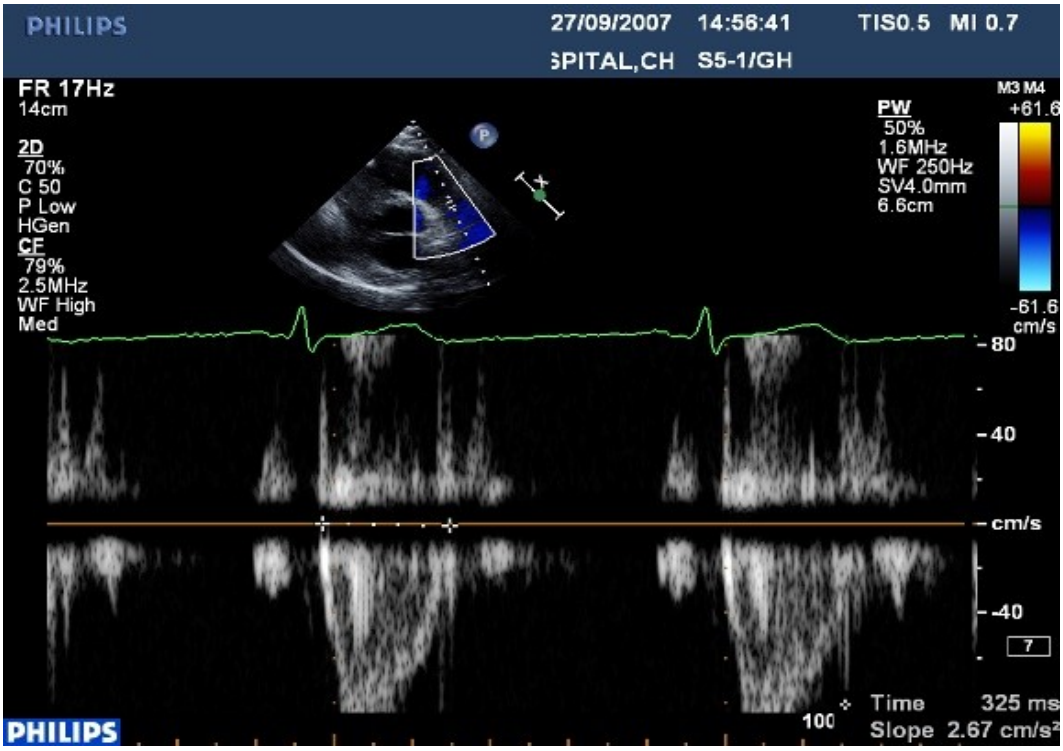


Picture 14. Measurement of MPI: Same patient as above. Value 'b' which is ET which is measured across the RVOT in parasternal short axis view.

MPI is calculated in this patient of IWMI with RVMI using the formula
MPI = a-b / b which is 417-271 / 271 = 0.54 in this patient

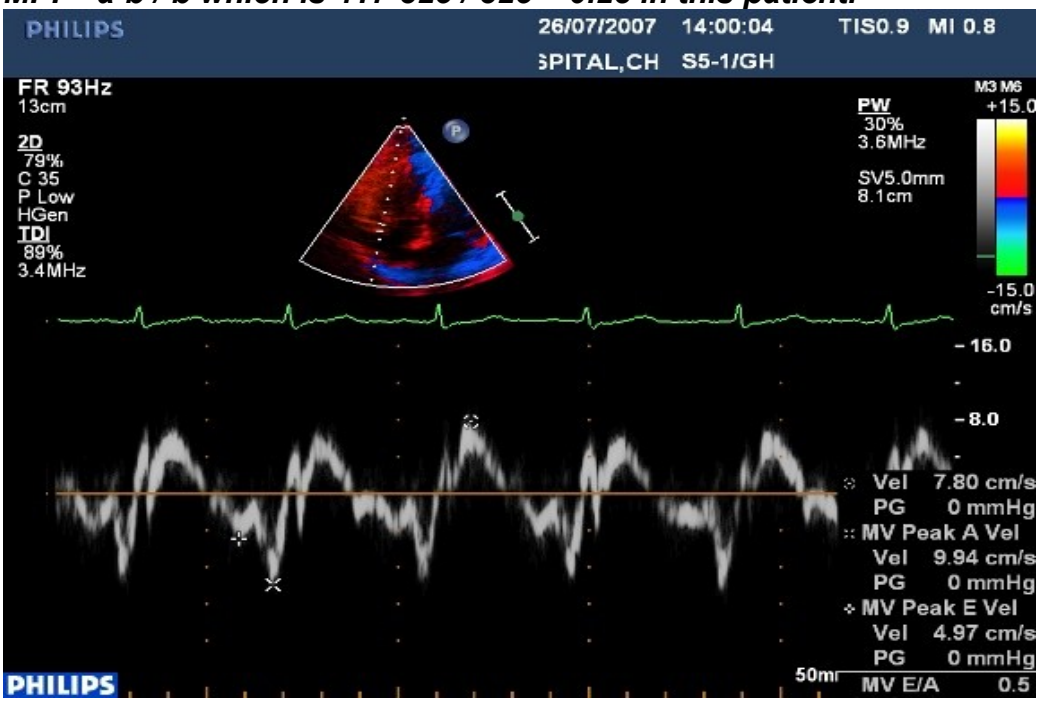


Picture 15. Measurement of MPI: Value 'a' which is IVCT + ET + IVRT in apical 4 chamber view- a patient of IWMI without RVMI

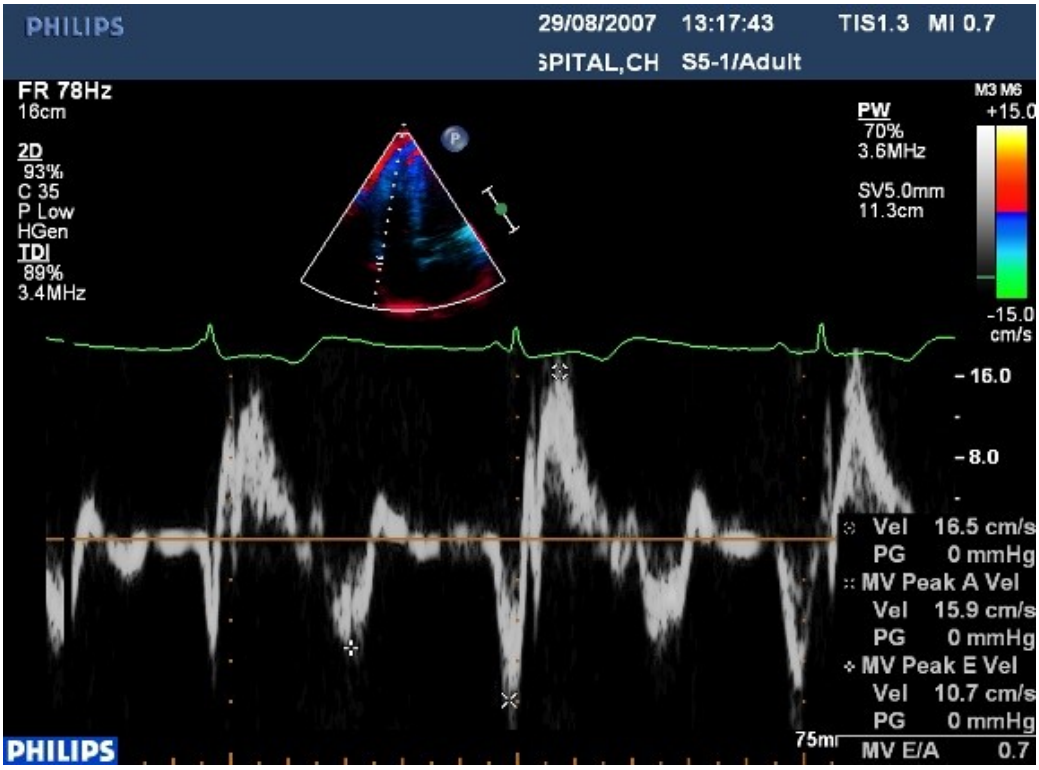


Picture 16. Measurement of MPI: Same patient as above. Value 'b' which is ET which is measured across the RVOT in parasternal short axis view.

MPI is calculated in this patient of IWMI without RVMI using the formula
MPI = a-b / b which is 417-325 / 325 = 0.28 in this patient.



Picture 17. TDI of RV lateral wall: A patient of IWMI with RVMI; Sm & Em are markedly reduced.



Picture 18. TDI of RV lateral wall: A patient of IWMI without RVMI; Sm & Em are normal.

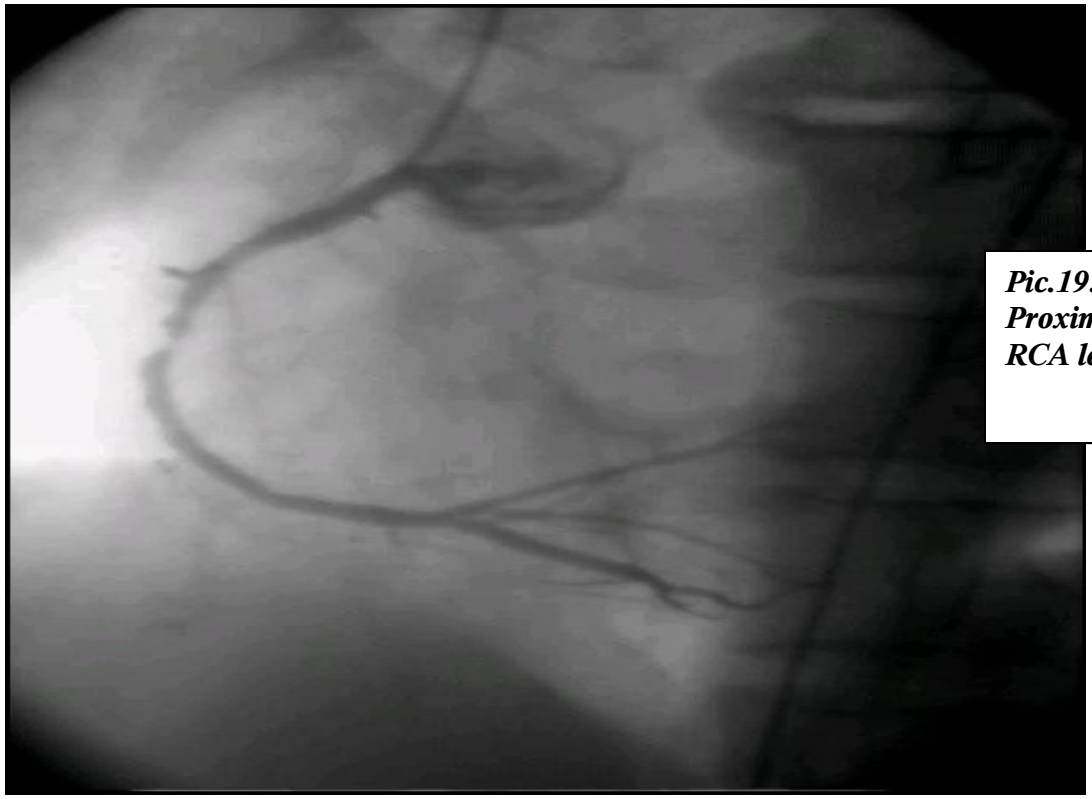
Coronary angiography:

Coronary angiography was done in 66 patients, of whom 28 showed right ventricular involvement by echocardiography. Out of the 66 patients,

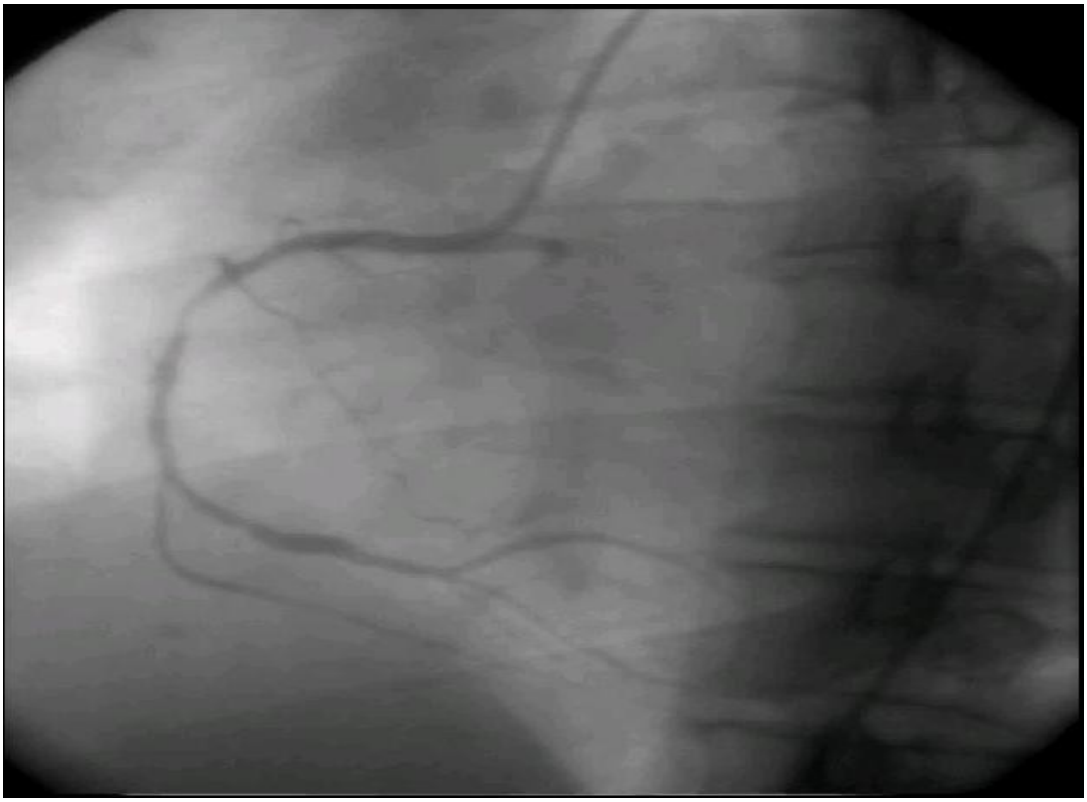
- Single vessel disease was found in 34 patients (Pic. 19 – 22)
- Double vessel disease found in 14 patients and
- Triple vessel disease in 18 patients.

Those who had two and three vessel disease were mostly diabetics and belonged to the >40 years age group; even in those people, the infarct related artery was the RCA or the LCX.

Pictures 19 & 20 are CAGs showing proximal RCA disease (with RVMI) and figures 21 & 22 are CAGs showing mid RCA lesions.

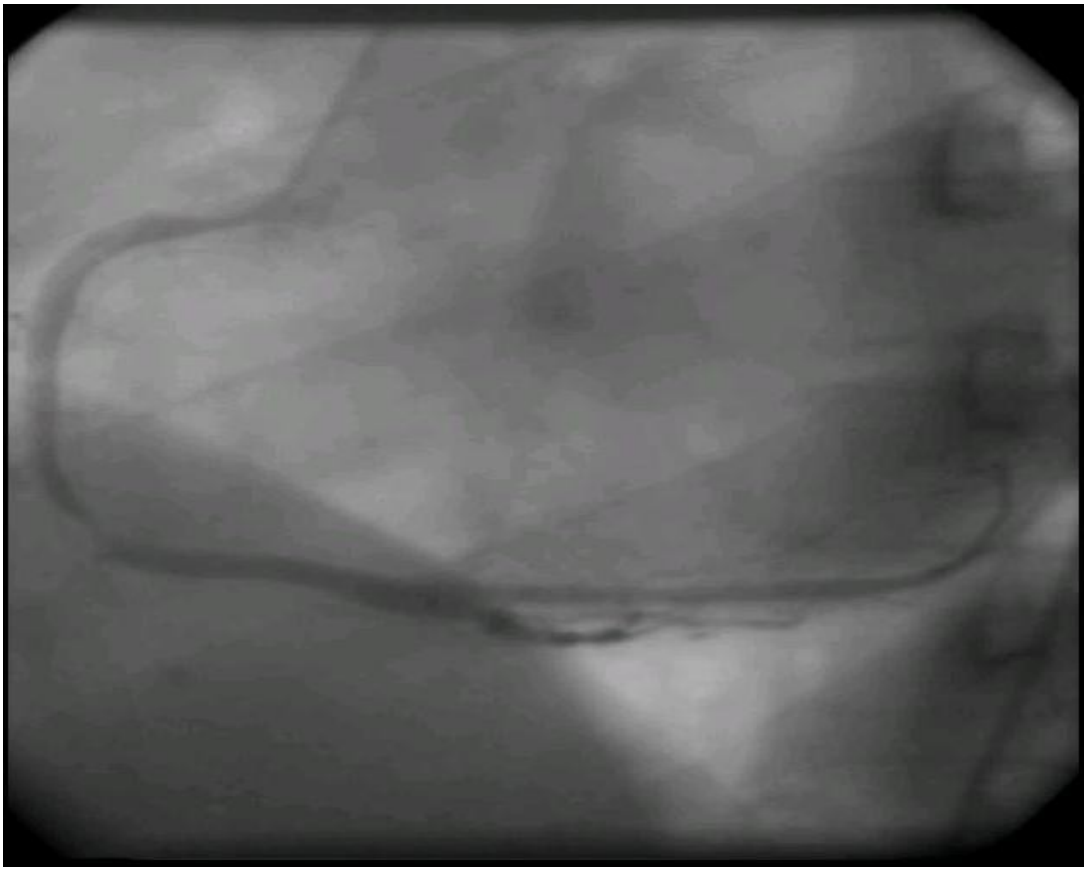


Pic.19: Proximal RCA lesion



Pic.20: Proximal RCA lesion

Pic.21: Mid RCA lesion



Pic.22: Mid RCA lesion

DISCUSSION

In patients with RVMI, the risk of death in the hospital is high and major complications are greater. Right ventricular infarction contributes markedly to hemodynamic instability, atrio-ventricular conduction blocks, and in-hospital mortality in patients with inferior wall myocardial infarction. Systolic right ventricular function is an important predictor in the course of myocardial infarction.

The poorer outcomes among patients with RVMI are not mediated entirely by RV dysfunction. Recent research points towards a new explanation for the higher complications even when the extent of RV infarction is small. Stimulation of RV vagal afferents by stretch leads to cardio-inhibitory Bezold-Jarish reflex resulting in higher than expected incidence of complications especially in the first few days after acute coronary event with RV involvement[41].

Gumina et al in Am J Cardiol in 2006[28] noted that, RVMI secondary to isolated right coronary artery (RCA) disease had a 10-year actuarial survival of 62%, versus 52% for those with combined RCA and left coronary artery (LCA) disease ($p = 0.21$). Mortality within the first year after infarction was substantial for all patients with RVMI; however, there was a nonsignificant trend for patients with RCA disease (18%) versus those with RCA and LCA disease (27%; $p = 0.21$). Occurrence of congestive heart failure, atrial fibrillation, and mechanical complications was similar in the 2 groups. In conclusion, RVMI is associated with substantial first-year mortality, which decreases to a much lower attrition rate between years 1 and 10, with no greater long-term mortality in those patients with concomitant LCA disease.

The clinical triad of hypotension, raised JVP and clear lung fields occurs in only 25% of individuals with right ventricular myocardial infarction[42].

The use of electrocardiography for the diagnosis of right ventricular infarction, which is the most common modality used, also lacks sensitivity and specificity and the changes are also transient in

nature. Hence, the need for other non-invasive modalities for the assessment of right ventricular function in the presence of right ventricular myocardial infarction.

Problems with Non-invasive Assessment of the Right Ventricle:

A number of factors contribute to the complexities of estimating right ventricular function. Whilst the left ventricular cavity approximates to an ellipsoid model in healthy individuals the right ventricle is considerably more complex. The main body of the chamber is crescentric and truncated with separate inflow and outflow portions. The outflow portion or infundibulum may account for up to 25% of the total right ventricular volume. The fact that the chamber poorly approximates to any convenient geometric model means that volume calculated with these models only crudely represents true volume. Marked regional differences exist in the extent of fibre shortening and contribution to stroke volume between different components of the right ventricular with the contraction–relaxation sequence of the inflow portion preceding that of the infundibulum[5]. The inaccessibility of the right ventricle behind the sternum often leads to inadequate image quality by conventional imaging modalities and this is particularly pertinent in patients with chronic pulmonary disease, a group in whom right ventricular dysfunction may be present. In addition, the problem of accurately locating the endocardial boundary of the anterior wall of the chamber is compounded by a variable trabeculation pattern with the apical component having much coarser trabeculations than the corresponding zone of the left ventricle. Chamber orientation varies considerably between patients, particularly in those with right ventricular pressure or volume overload. In addition to myocardial function the shape and performance of the right ventricle depends on extrinsic factors such as pre-load, afterload and left ventricular performance. A limitation of conventional imaging methods in clinical practice is that these factors are frequently disregarded.

Clinical implications of RVMI:

The severity of the hemodynamic abnormalities associated with RV infarction is related to the extent of RV ischemia and consequent RV dysfunction as well as to the restraining effect of the

pericardium, LV function, and ventricular interdependence. It has now been shown both experimentally and clinically that the intact LV may assist RV ejection by LV septal contraction causing a bulging into the RV which generates an active RV systolic pressure wave and systolic force sufficient for pulmonary perfusion. Loss of this mechanism with concomitant LV infarction, particularly when the interventricular septum is involved, may lead to further hemodynamic deterioration in patients with RV infarction. Furthermore, augmented atrial contraction is necessary to overcome the stiffness of the ischemic RV, and factors that impair RV filling (intravascular volume depletion, concomitant atrial infarction, and loss of atrioventricular synchrony) may severely compromise hemodynamics and result in cardiogenic shock. In comparison to the LV, the RV is poorly adapted to compensate for the increase in afterload, with its large surface area and thin free wall, and this may explain the rapid hemodynamic compromise and earlier onset of hypotension and shock in patients with predominant RV shock. Right ventricular infarction has been associated with both occlusions of a non-dominant RCA and significant RV hypertrophy that increases the susceptibility of the RV to ischemia[39]. It is unclear why all proximal RCA occlusions are not associated with RV infarction, but the lower oxygen requirement of the RV, increased coronary blood flow during systole, increased collateral flow from the left coronary artery, and diffusion of oxygen from intra-cavitary blood through the thin wall of the RV have been implicated.

RVMI recovers fast and suffers less ischemic insult because:

- The right ventricle has lesser muscle mass and thickness, hence the infarct is not huge
- The coronary perfusion through the Right Coronary artery occurs in both systoly and diastoly.
- There is greater amount of collateral flow to the right ventricle compared to occlusion of the left system due to trans-coronary perfusion gradients during RCA occlusions[43].
- There is direct diffusion of oxygen from the right ventricular cavity[44].

Treatment of RVMI:

Although studies differ concerning the influence of RV infarction on long-term prognosis, it is

clear that in the majority of survivors, the clinical (and echocardiographic/radionuclide)[40] manifestations of RV dysfunction return to normal. Optimal management aimed at support of the RV and reversal of RV ischemia is essential and currently consists of maintenance of adequate RV preload with volume loading (although excess volume further compromises RV function), preservation of RV synchrony, reduction in RV afterload (particularly when LV dysfunction is present), and inotropic support of the RV. Several studies also suggest a role for early reperfusion with fibrinolytic therapy or primary angioplasty. Furthermore, early recognition of predominant RV infarction in the pathogenesis of cardiogenic shock is critically important to ensure not only that appropriate treatment is instituted but that therapies that may be problematic (nitrates, morphine) are avoided.

Future directions:

Three Dimensional Echocardiography:

The advent of three-dimensional echocardiographic reconstruction removes the longstanding limitation imposed by standard imaging planes. Effectively, it eliminates the need for geometric assumptions of the complex right ventricular architecture and allows improved endocardial detection. The feasibility and reliability of three-dimensional echocardiographic imaging has been examined by in vitro and in vivo[14] studies, and modern data collection and gating systems minimize artefactual errors due to subject motion or respiration. Three-dimensional echocardiographic measurements of right ventricular volumes correlate closely with those of magnetic resonance imaging and permit reliable serial measurements. The accuracy and reproducibility of the technique improve the evaluation of right ventricular dysfunction. While three-dimensional echocardiography appears to be excellent for objective and accurate measurements of right ventricular geometry there is a paucity of information on its prognostic value, it is currently not widely available for routine use and time is required for post-acquisition reconstruction of the images.

Automated border detection methods:

They have been employed in the assessment of global right ventricular function in patients with a variety of underlying pathologies[15]. Estimation of changes in right ventricular dimensions based on this technique is in close agreement with contrast angiographic data. Colour kinesis can be used to quantitatively assess segmental right ventricular function and has been shown to be of value in a variety of pathologies affecting the right ventricle with excellent correlation between ventriculographic and colour kinesis measurements. Contrast echocardiography has been applied to the right ventricle in an attempt to overcome difficulties with endocardial border definition. In healthy subjects the correlation between echocardiographic and radionuclide derived right ventricular ejection fraction significantly increased and inter-observer variability improved with the addition of contrast. [16] Tricuspid annular motion can be assessed easily using Doppler tissue imaging[17]. An advantage is that measurement is independent of geometric assumptions and endocardial border tracing. This method has been accepted as a convenient means of quantitatively evaluating right ventricular systolic function and has been used as a diagnostic tool by a number of groups. The sensitivity of tricuspid annular motion for the detection of early right ventricular dysfunction may be superior to more conventional imaging techniques and a recent study has suggested that reduced systolic annular velocity is highly predictive of right ventricular dysfunction as measured by radionuclide ventriculography[18].

Intra-cardiac echocardiography:

Intra-cardiac echocardiography is able to accurately delineate the entire right ventricular architecture in the invasive catheterization laboratory setting. Using a sequential catheter pull-back technique it has been demonstrated that right ventricular volume and function could be accurately assessed compared to a directly measured standard model[19].

Strain & strain rate imaging:

The principal advantage of strain rate and strain against velocity measurements is that overall heart motion, cardiac rotation, and contraction in adjacent segments is accounted for with strain imaging. However, there has been, so far, no systematic investigation testing strain and strain rate, as well as

systolic RV free wall velocity for the characterisation of systolic RV function.

Comparison with other studies:

Right ventricular cavity size by echocardiography:

In a study by Dokainish et al [24] published in 2005, Right ventricular end-diastolic & end-systolic dimensions were measured in 50 patients within 48 hours of a first attack of IWMI. The end-diastolic dimension was 41±5mm in those with right ventricular myocardial infarction and it was 36±4mm in those without right ventricular involvement and it had statistical significance.

Garcia-Fernandez et al[25] measured right ventricular dimensions in right ventricular myocardial infarction and established that diastolic dimension greater than 8mm/m2 is highly indicative of ischemic right ventricular dysfunction, provided that other causes of right ventricular dilatation, but the sensitivity of this findings is low, about 50%, the same is true for a RVDD/LVDD ratio greater than 0.63.

Anna Vitoria et al[26] in 2000 noted that all patients with RVI showed a right ventricular dilation (LVEDD 3.2 ± 1.3 cm) with increased right ventricular end diastolic and end systolic areas. The interventricular septum had an abnormal motion in 86% of the patients.

In our study, the RV end diastolic diameter was 32 ± 13 in patients with RVMI, compared with a value of 20 ± 14 in patients without right ventricular involvement.

Right ventricular contraction abnormalities:

In the same study by Dokainish et al[24], right ventricular contraction abnormalities was present in 11/22 patients (60%) with right ventricular myocardial infarction and only in 6/28 (20%) patients without right ventricular involvement and it had statistical significance with p=<0.02.

Garcia-Fernandez et al[25] concluded that the most common site of involvement is confined to the posterior segment; in 30% there is also abnormal contraction of the lateral wall and 10% of the cases present akinesis of the anterior, lateral and posterior segments. Those patients with the most severe

right ventricular dysfunction presented a higher number of right ventricular wall segments with abnormal wall motion. Paradoxical septal motion is a common finding after right ventricular infarction and has been attributed to volume overload and alterations in right ventricular compliance, near of 50% present abnormalities of septal motion and those patients with most severe ventricular dysfunction presented most frequently abnormal septal motion.

In our study, RV contraction abnormalities and IV septal motion abnormalities were noted in 41% of patients with RVMI, and only in 3% of patients without it. Significantly, the wall motion abnormalities were noted in those patients who had their echo done within 24 hours of symptom onset.

Tricuspid regurgitation jet:

Anna Vittoria et al found that tricuspid regurgitation was detected in 26 out of 44 Patients with right ventricular myocardial infarction. The mean peak velocity of tricuspid regurgitation was 3.8 ± 0.8 m/s[26].

In our study, tricuspid regurgitation was noted in 21/44 (48%) patients with right ventricular involvement; this regurgitation, when quantified by colour doppler, was mild in a majority of cases, moderate in some and severe in none.

The pulmonary artery systolic pressure and thence the right ventricular systolic pressure was slightly elevated and above normal in patients of Group 1, compared to Group 2.

Tricuspid annular peak systolic excursion (TAPSE):

In a study conducted by Alam et al[27], from the echocardiographic apical 4-chamber views, the systolic motion of the tricuspid annulus was recorded at the RV free wall with the use of 2-dimensional guided M-mode recordings. The tricuspid annular motion was reduced in inferior Ml compared with that in healthy individuals (20.5 and 25 mm, P <.001). The peak systolic velocity of the tricuspid annulus was significantly reduced in inferior Ml compared with that in healthy individuals (12 vs 14.5

cm/s, P<.001) and patients with anterior MI (12 and 14.5 cm/s, P<.001).

Meluzin et al found that there was a good correlation between systolic annular velocity and right ventricular ejection fraction (r=0·648, P<0·001). A systolic annular velocity <11·5cm(s–1) predicted right ventricular dysfunction (ejection fraction <45%) with a sensitivity of 90% and a specificity of 85%.

afterload and contractility.

In our study, the tricuspid annular excursion was significantly decreased in patients with right ventricular myocardial infarction (14.1 ± 2.4mm Vs. 18.2 ± 1.9), signifying that RV systolic function was depressed in those patients

Myocardial performance index (Tei index):

Tei and coworkers suggested an easily obtainable Doppler-derived index of right ventricular dysfunction, which combined elements of systolic and diastolic function. These authors measured the index in a population of patients with primary pulmonary hypertension and compared the results with those obtained in a normal population. Tei and colleagues found a significant correlation among the index, clinical symptoms, and overall survival, supporting the hypothesis that the index correlates with the clinical severity of right ventricular dysfunction. The right ventricular MPI is an interesting parameter for the early diagnosis of right ventricular involvement during acute myocardial infarction. The index increased during hospitalization, whichoccurred from 3 to 5 h after the onset of chest pain. A RV MPI values of >0.65 is associated with an increased mortality[26].

Alagesan R et al[37] in their study of South Indian patients with inferior wall myocardial infarction found that RV MPI was significantly elevated (from baseline of 0.20±0.05 in controls) to a mean of 0.53±0.22 in RVMI patients (p<0.001). IWMI did not elevate MPI significantly (0.21±0.17, p=NS). RV MPI >0.30 has a high sensitivity (82%) and specificity (95%) for the diagnosis of RVMI in the presence of acute IWMI.

AV Mattioli MD[26] et al in her study found that isovolumetric contraction times (136 ± 30 versus 49 ± 11 ms; $P < 0.01$), and relaxation times (71 ± 28 versus 37 ± 9 ms; $P < 0.01$) were prolonged in patients with right ventricular infarction, whereas the ejection time was significantly reduced (250 ± 31 versus 330 ± 26 ms; $P < 0.001$). The myocardial performance index was significantly increased in patients with right ventricular infarction (0.85 ± 0.2 versus 0.26 ± 0.1; $P < 0.01$).

Ozdemir et al[36] found that a right ventricular MPI value of >0.7 was 94% sensitive and 80% specific for the diagnosis of right ventricular dysfunction.

In our study, Myocardial performance index was found to be statistically significant, it was 0.45 ± 0.07 in patients with RVMI, compared to near normal values of 0.29 ± 0.04 in patients without right ventricular infarction.

We evaluated the Tei index in patients with RVI, and we suggest that the index offers additional information on right ventricular performance in patients in the coronary care unit. We believe that this index should be measured in all patients with inferior LVI and possible involvement of right ventricle.

Tissue Doppler Imaging:

In an extensive adult patient population, a Swiss study[22] demonstrated that TDI of the systolic lateral tricuspid annular long axis velocity (TVlat) is accurate to characterize systolic RV function independent of most, pathophysiologically meaningful cofactors. A velocity of 12 and 9 cm/s differentiates among normal and moderately reduced RV ejection fraction, respectively between moderately and severely impaired RV EF.

Since it is only above 40 mm Hg that pulmonary artery pressures impacts on TVlat, it affects the relationship between TVlat and RV EF especially in patients with pure PAHT in whom the former cannot be used for RV function assessment. With regard to the specific TVlat threshold value of 12 cm/ s for the distinction between normal and impaired RV function in our investigation, the study by Oezdemir[21] and coworkers using RV TDI has to be cited, because it found an identical cut-off with almost identical sensitivity and specificity of 81 and 82% for the diagnosis of RV myocardial

infarction. The limitations of TDI is that it represents myocardial motion only in the longitudinal direction, and not in the circumferential plane.

Furthermore, on the basis of the correlation between isovolumic contraction velocity at basal segment and right atrial mean pressure, we tested the sensitivity and specificity of a cut off value below 6 cm/s indicating an increased right atrial pressure (>6mmHg). The sensitivity and specificity were 100% and 78%, respectively[29]. These findings may be of importance as disturbance of myocardial motion occurs predominantly during the isovolumic phases (i.e. contraction and / or relaxation), and may therefore be a sensitive marker for myocardial dysfunction.

Alam et al[27] in his study from Sweden, patients with RV infarction had a significantly decreased peak systolic tricuspid annular velocity (13.3 and 10.3 cm/s, P <.001) and peak early diastolic velocity (13 and 8.2 cm/s, P<.001) when compared with patients with inferior wall myocardial infarction without RV involvement.

Mustafa Yilmaz et al[34] in his study of forty-eight patients with acute inferior myocardial infarction and 24 age- and sex-matched healthy controls were included in this study. Twenty-four patients had electrocardiographic signs of inferior myocardial infarction without right ventricular infarction (group I), and the other 24 patients had electrocardiographic signs of inferior myocardial infarction with right ventricular infarction (group II). From the echocardiographic apical four-chamber view, peak systolic, early diastolic, and late diastolic velocities of the tricuspid annulus at the right ventricular free wall were recorded with the use of pulsed-wave Doppler tissue imaging. The tricuspid annular peak tissue Doppler imaging systolic velocity was significantly lower in group I (14.03 ± 2.57 cm/s, $P < 0.005$) and in group II (8.50 ± 0.84 cm/s, $P < 0.005$) than in controls (16.63 ± 2.31 cm/s). The tricuspid annular peak systolic (8.50 ± 0.84 cm/s vs 16.63 ± 2.31 cm/s) and peak early diastolic (10.99 ± 3.28 cm/s vs 19.39 ± 4.3 cm/s) velocities were significantly lower in group II than in group I, as compared with controls ($P < 0.001$). Peak early diastolic velocity of tricuspid annulus (10.99 ± 3.28 cm/s vs 19.39 ± 4.3 cm/s) was significantly lower in group I than in controls ($P < 0.001$); however, late

diastolic velocity was significantly lower in group II (15.98 ± 5.08 cm/s, P <0.05) than in group I (18.21 ± 2.63 cm/s, P < 0.05) and in controls (19.02 ± 5.29 cm/s). The results of this study indicate that tricuspid annular peak systolic and early diastolic velocities are reduced in patients with right ventricular infarction. The velocity of the tricuspid annulus by tissue Doppler imaging is simple and can be used to distinguish whether patients with inferior myocardial infarction have right ventricular infarction.

Oguzhan et al[35] on 2004 studied 35 patients with inferior wall myocardial infarction, 14 of whom had right ventricular infarction and found that Systolic and early diastolic velocities at the lateral tricuspid annulus were significantly reduced in patients with inferior MI with RV infarction compared with those in healthy individuals (7.8 ± 1 vs. 11 ± 2 cm/s, p < 0.002) and patients with inferior MI without RV infarction (7.8 ± 1 vs. 10 ± 1 cm/s, p < 0.002). The late diastolic lateral annular velocity did not differ between the groups. Systolic and early diastolic RV free wall velocities were also significantly decreased in patients with RV infarction compared with those in healthy individuals (7 ± 1 vs. 8.7 ± 1 cm/s, p < 0.01; 6.3 ± 2 vs. 8.7 ± 2 cm/s, p < 0.05, respectively) and patients with inferior MI without RV infarction (7 ± 1 vs. 9 ± 2 cm/s, p < 0.01; 6.3 ± 2 vs. 8.3 ± 2 cm/s, p < 0.05, respectively).

In our study, it was found that the right ventricular free wall Sm and Em values were statistically significantly depressed in patients with right ventricular myocardial infarction compared to those without RV involvement. (Sm 9.4 ± 0.7 Vs. 12.1 ± 1.1; Em 6.8 ± 0.7 Vs. 8.4 ± 1.0). However, the RV free wall late diastolic velocities (Am) and the septal velocities did not differ much in patients with and without right ventricular myocardial infarction (Am 7.4 ± 1.1 Vs. 8.6 ± 1.2; Septal velocities 6.4 ± 0.9 Vs. 6.2 ± 0.8).

ECG, Echo & CAG correlation:

- Echo identified RVMI in 9 patients who did not show RVMI on ECG, and this was confirmed by CAG
- 11 patients with RVMI ECG did not have evidence of RVMI on echo as well as on CAG.

CONCLUSIONS

- On tissue doppler imaging, right ventricular free wall systolic velocity and early diastolic velocity in patients with RVMI was less compared to patients without RVMI and the correlation was statistically significant.

- Right ventricular free wall late diastolic velocity and septal velocities did not correlate well with RV dysfunction.

- Right ventricular myocardial performance index (Tei index) correlated with RV dysfunction and attained statistical significance. Increased MPI values were also associated with higher mortality.

- Right ventricular dimension & regional wall motion abnormalities were insignificant in detecting RVMI.

- RV dysfunction was worse in those whose echo was done within 24 hrs, when compared with those done after, showing that RV function improves with time.

- Thrombolysed patients had lower Myocardial performance index values, compared to those who were not thrombolysed.

- Age and sex did not significantly correlate with right ventricular function after a myocardial infarction.

- Echo can identify right ventricular dysfunction in patients who have no or doubtful findings of RVMI on ECG.

LIMITATIONS OF THE STUDY:

1. Analysis of LV MPI was not part of our study. This may also significantly contribute to overall hemodynamics in RV MPI assessment in RVMI.
2. Myocardial velocities obtained by TDI in the apical 4 chamber reflect the movements of the myocardium only along the long axis. The contraction of the ventricular circumferential fibres does affect the TDI.
3. Tricuspid annular movements do not represent the function of the entire RV myocardium.
4. Even though echocardiography was performed within the first 48 hours after IWMI, it is possible that some patients may have already recovered form the damage in the right ventricle.
5. Magnetic resonance imaging which is the gold standard for right ventricular function was not done in our cases, due to financial constraints.
6. There is no postmortem detailing of pathology among the deaths.

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GLOSSARY AND ACRONYMS

AWMI- Anterior Wall Myocardial Infarction.

IWMI- Inferior Wall Myocardial Infarction.

RVMI- Right Ventricular Myocardial Infarction.

LWMI- Lateral Wall Myocardial Infarction.

CAD- Coronary Artery Disease.

TTE- Trans Thoracic Echocardiogram.

MRI- Magnetic Resonance Imaging.

STEMI- ST segment Elevation Myocardial Infarction.

LVEF- Left Ventricular Ejection Fraction.

RV- Right ventricle; LV-Left ventricle.

EDD – End Diastolic Diameter

MPI – Myocardial Performance Index

EF – Ejection Fraction

ECG - Electrocardiography

IVCT – Isovolumetric Contraction Time

IVRT – Isovolumetric Relaxation Time

TDI – Tissue Doppler Imaging

TAM – Tricuspid Annular Movement

TAPSE - Tricuspid Annular Peak Systolic Excursion

Sm – Myocardial systolic velocity

Em – Early phase of diastolic myocardial velocity

Am – Late phase of diastolic myocardial velocity